HEMODYNAMIC CORRELATES OF XYZ

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1975

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A THESIS SUBMITTED IN PARTIAL
FULFILLMENT OF THE REQUIREMENTS FOR
THE DEGREE DOCTOR OF MEDICINE

YALE UNIVERSITY
NEW HAVEN, CONNECTICUT
MARCH 1975

To My Family

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Acknowledgments:

To Doctor Norman S. Talner for his invaluable support and deep felt friendship and for illuminating with towering brilliance and originality insights into cardiopulmonary physiology which come from his inspiring ability to think about patients with clarity, excellent judgment and profundity

To Doctors Berman, Browne, Fish, Nudel and the other attendings, trainees and staff in Pediatric Cardiology who contributed most often after hours with their philosophy, their fellowship and their knowledge and experience in caring for the patients in this study

And to Doctor Genel and the Pediatric Clinical Research Center for use of their Wang 600 computer.

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Predictability was a fundamental goal of classical science, wherein Nature unraveled herself in experiments of straightforward casuality. In the modern world, where man's vision is sharpened in the astonishment of unexpected happenings, what is valuable is the capacity to understand change and coincidence rather than the power to divine certainty. Electrocardiographers have always sought to predict the hemodynamic state from the voltage and orientation patterns of an electrocardiogram and failed. Yet, as practical philosophers, they realize that the relationship between abnormalities on the electrocardiogram and in the structure of the heart is more than mere coincidence and possesses great meaning.

In the past fifteen years, studies on corrected electrocardiographic lead systems, principally that of Frank (1956), in which there is a simultaneous display of corrected orthogonal voltages on the right-left or x axis, the superior-inferior or y axis and the anterior-posterior or z axis, have suggested excellent correlations between hemodynamic parameters in patients with known cardiac disease and the magnitude of voltages calculated from tracings taken with corrected electrocardiographic lead systems (Gamboa & Hugenholtz, 1964; Ellison & Restiaux, 1972). In contrast, reported correlations between hemodynamic parameters and the amplitude of voltages calculated from the standard twelve lead electrocardiogram (ECG) have not been good (Cayler et al, 1958; Braunwald et al, 1963). Although the ability to assess overload conditions

of the right and left ventricles by a non-invasive technique such as the electrocardiograph is extremely worth developing, there have been few studies in which the XYZ electrocardiogram was used to identify or estimate the severity of ventricular pressure or volume overloading in pediatric patients of unknown cardiac status (Blumenschein et al, 1972).

In this study, the basis for using XYZ or the Frank scalar electrocardiogram in identifying children with heart lesions involving an overloaded ventricle is reviewed. The reasons why a congenital heart defect may cause changes in normal electrocardiographic patterns are presented and followed by a discussion of the standard twelve lead electrocardiogram, standard ECG criteria for hypertrophy, and the advantages and disadvantages of the XYZ when compared to the ECG.

With this background, three sets of XYZ criteria incorporating the voltage and orientation requirements used in the ECG criteria for hypertrophy were designed to identify increased ventricular forces. The diagnostic accuracy of these XYZ criteria was compared with that of the standard twelve lead ECG in seventy-four patients who subsequently underwent cardiac catheterization. Correlations between the magnitude of the XYZ voltages and the amount of pressure or volume overloading are presented.

I. REVIEW: XYZ in the Diagnosis of Pressure & Volume Overloaded Ventricles

A. Ventricular Hypertrophy as a Consequence of Congenital Heart Lesions

The consequence of a congenital heart lesion is a disruption of the normal pattern of blood flow through the heart. Depending on the nature of the changes in cardiac anatomy, the result can be a volume or pressure load on the ventricle. One can describe the severity of the overload by using two hemodynamic parameters: the peak systolic intraventricular pressure and Qp/Qs--- the ratio of the pulmonary to systemic blood flow--- which quantifies the amount of shunting to or overload on a ventricle. For example, aortic stenosis represents a pressure overload on the left side of the heart and the peak systolic intraventricular pressure in the left ventricle can be significantly elevated. On the other hand, the classic example of a volume overload of the right ventricle is the lesion of isolated atrial septal defect, where Qp/Qs is greater than the normal value of 1.

Although there are other adaptive mechanisms by which the heart can respond to chronic excessive loading, such as utilization of the Frank-Starling mechanism or increase in the contractile state by way of the cardiac adrenergic nervous system (Spann, 1969), the principal adaptive mechanism to be considered here is an increase in contractile element mass or ventricular hypertrophy. In a pressure overload such as aortic stenosis, the left ventricle responds with concentric hypertrophy and relatively little or no dilatation. Although

the contractile force across the total cross sectional area of myocardium $T = P(pi)r^2$* is increased, the stress per unit area of muscle wall $T = Pr/2delta$* is maintained at a normal level. In a volume overload such as aortic insufficiency, there is dilatation as well as eccentric hypertrophy. The advantage of the dilated heart is that each fiber shortens less to eject a given volume of blood. Another consequence of dilatation is that the fibers in the wall of the ventricle must develop a greater contractile tension $T=Pr/2$,* the force per unit length of circumference and the entire thickness of myocardial wall, than is necessary in the normal heart to generate a given pressure. This requirement of additional tension can itself lead to hypertrophy.

I.B. Relationship Between Ventricular Hypertrophy and Electrocardiographic Potentials

1. Effect of Hypertrophy on Electrocardiographic Potentials

Just as the pattern of ventricular hypertrophy is thought to reflect the nature of the original cardiac lesion, so electrocardiographic potentials are believed to indicate

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*Each of these formulae is based on the law of LaPlace applying to a membrane which encloses a volume of spherical or cylindrical shape(Badeer, 1963). According to LaPlace's Law, as the radius of the volume of the heart increases, more tension must be developed by each fiber to produce or maintain a given intraventricular pressure. r=average radius of the ventricle, assuming a spherical or cylindrical shape. P=transmural pressure across the wall of the ventricle. delta=ventricular wall thickness. pi=3.1416....

the pattern of myocardial geometry. In a study which compared the activation sequence of the right ventricular epicardial surface in patients with a normal right ventricle to those with congenital heart lesions associated with right ventricular hypertrophy, Boineau (1968) concluded that if it is possible to assume that alterations in the Purkinje network do not contribute to changes observed on the epicardial surface of the heart, "the degree of delay and changes in the sequence of activity appear to be related to the extent and to the symmetry of the hypertrophy."

To elaborate, Boineau found that in patients with pulmonic stenosis, where the hypertrophy is relatively concentric (hypertrophy is less remarkable in the inflow tract of the right ventricle and extends up to the pulmonary valve), the delay in epicardial excitation was uniform at all points. The sequence of activation recorded from the surface of the right ventricle did not differ from that of the normal heart. In contrast, in patients with Tetralogy of Fallot, where the hypertrophy was markedly assymetric, activation began and ended at different regions than in the normal right ventricle. While the free wall overlying the region between the infundibular stenosis and the pulmonary valve was essentially normal in thickness, the lateral and posterior portions of the right ventricle overlying the inflow tract were markedly hypertrophied and activated late.

The mechanism by which thickening or other changes in the geometry of the ventricular wall affect the electric field is thought to be via changes in the properties of the entire myocardium acting as an electrical matrix rather than changes in the number or properties of single cell generators. This is suggested by the work of Linzbach(1960), who showed that increases in mass in concentric hypertrophy were the result of increases in cell volume rather than number and the work of Uhley (1961), who found the transmembrane potentials of individual hypertrophied cardiac muscles to be normal.

When hypertrophy occurs, there can be measurable differences in the electrical wavefront without changes in the density of dipoles per unit area of myocardium (Boineau, 1968). Mere lack of symmetry in the hypertrophic process or non-uniform distribution of the cardiac conduction system can alter electrocardiographic forces significantly. When there is a stimulus to hypertrophy, increases in cardiac mass can occur only where there is room within the thorax and within the restraints laid by fixed connections to the systemic and pulmonary circulations. Growth in areas which are plentifully supplied by the Purkinje fibers are likely to be less significant electrocardiographically than areas which are not supplied by dense Purkinje fibers and which register increased electrical fields on the electrocardiogram. The outer wall of the ventricle is not only known to hypertrophy more in porportion to the endocardium but also to be electrically more active. Waves of excitation are known to travel much faster along endocardium than epicardium and thus to be more silent electrocardiographically.

Hypertrophy of any local area of myocardium results in a delay in activation; the resultant asynchrony unmasks and alters the electrocardiographic forces.

In addition, alterations in the cardiac anatomy influence the amount of blood within the chambers of the heart, which also is thought to influence the height and configuation of the electrocardiographic potentials. According to the recent review by Voukydis(1974), myocardial excitation in a radial direction towards the blood mass is associated with an increasein the magnitude of the electrical potential when the volume of blood is increased. But tangential myocardial excitation is associated with a decrease in the magnitude of the potential when the volume of blood is increased. This theory is consistent with the finding of peaks in the time plot of the strength of the instantaneous vector which represents cardiac depolarization .
The quantification and further understanding of these correlations will enable one to know precisely how much of a contribution tissue hypertrophy makes in the genesis of the electrocardiographic tracing.

Electrocardiographers have developed a large body of theory to explain and correlate events seen on an electrocardiogram with actual events in the depolarization of the heart.
The oversimplified but working principle of electrocardiographic interpretation is that leftward and posterior forces represent the depolarization of the left ventricle, which lies to the left of and behind the right ventricle, while the

rightward and anterior lying right ventricle is represented by rightward and anterior forces on the electrocardiogram.

It is now generally believed (Sodi-Pallares, 1970) that depolarization of the normal cardiac ventricles begins in the middle third of the left side of the septum and proceeds from the left side towards the right side. The septal or q vector is thus directed rightward, downwards and anteriorly and usually has a duration of .01 second. The concept of the septal q vector is firmly established, although Grant (1953) doubted that the q represented septal activation because he found the mean direction of the mean cardiac vector in the first .02 seconds of the QRS to be more tangential than perpendicular to the lie of the septum.

The next phase of the QRS wave represents the depolarization of the free walls of the right and left ventricles. In the first three years of life, the right heart can be normally dominant. But after this stage, the direction of depolarization should be leftward, posterior and inferior or superior because the left ventricle has become the dominant ventricle and the electrocardiographic potential of its free wall is ten to fifteen times stronger than that of the free wall of the right ventricle.

The terminal QRS forces tend to be directed posteriorly and somewhat rightward and represent the continuing depolarization of both the free wall of the left ventricle and the basal portion of the right ventricle. Although it is not possible to identify and measure the separate contributions made by depolarization of the right basal and left ventricular free wall to the electrocardiogram, Sodi-Pallares has suggested that the right basal wall vector has a

magnitude similar to that of the septal vector and is directed rightward, superiorly, and posteriorly.

I. B. 2. Development of ECG Criteria for Increased Ventricular Forces in Pediatric Patients

The development of pediatric ECG criteria for increased ventricular forces began once it was realized that pediatric patients have a normal morphologic pattern that is different from that of adults. In 1908, when the first study of the electrocardiogram in infants and children was published, in German by Nicolai and Funaro (Ziegler, 1951), the finding of a prominent S wave in lead I in many of the clinically normal children was unexplainable, since the association between right axis deviation and right ventricular hypertrophy in adult cardiac disease had already become well established. Not until 1913 did Lewis conclude from clinical studies that normal infants usually showed right axis deviation of the mean cardiac vector at birth and did not evolve towards a leftward or adult axis until later.

The development of unipolar leads during the 1930's enabled one to study in greater detail the transition of the QRS wave from right to left as well as T wave changes from infancy to childhood. Electrocardiographers began to realize that ECG's had to be interpreted differently, according to the anatomy and, hence, age of the patient.

The modern era of ECG criteria for ventricular hypertrophy in infants and children, as well as adults, involving the use of the precordial leads, dates back to the signal

papers of Sokolow and Lyon published in 1949 and of Myers et al published in 1948. These papers took the various electrocardiographic configurations of diagnostic importance being studied in research electrocardiographic laboratories and turned them into practical, ready-to-use guideposts for clinical diagnosis.

The papers by Sokolow and Lyon list all of the various electrocardiographic morphologic entities which are looked for in an electrocardiogram at the present time to support or exclude the electrocardiographic diagnosis of ventricular hypertrophy: abnormally increased amplitudes of R and S waves, reversed R/S ratios, degree of axis deviation of the mean cardiac vector, T wave inversion, small q in the far left or right precordial leads, increased QRS duration, delay in the ventricular activation time, signs of bundle branch block and other simulants of hypertrophy which exclude the diagnosis of ventricular hypertrophy.

Since then, numerous electrocardiographers, including Grant and Sodi-Pallares, have designed elaborate theories to explain every wave in the electrocardiogram. For example, Grant suggested that the first .01 to .02 seconds after the onset of ventricular activation provided information only about the septal regions, while the latter half of the QRS wave provided information only about the free walls of both ventricles. When there was involvement of both the early and late parts of the QRS wave, Grant felt that this was a sign of concentric hypertrophy, which is associated with pressure, as opposed to volume, overloads. When there

was involvement of only the latter part of the QRS interval, this was thought to represent eccentric hypertrophy, which is associated with a volume overload. Furthermore, Grant believed it was possible to specify not only the type of overload, but also which ventricle was being overloaded by determining from the electrocardiogram which ventricle showed increased forces.

Generally, the authors of ECG criteria have not been as optimistic about the possibility of distinguishing pressure from volume overloads as they are about identifying the presence of either right or left ventricular hypertrophy. Changes in the magnitude and orientation of the electrocardiographic forces representing ventricular depolarization are most useful for the diagnosis of hypertrophy (Sokolow and Lyon, 1949; Kjellberg et al, 1954; Gasul et al, 1966; Guntheroth, 1965; Keith et al , 1967; Liebman, 1968; Krovetz et al, 1969; and Nadas , 1972). Other criteria, such as delay in the ventricular activation time, QRS duration and RST-T changes, are either too non-specific or have less basis for their use than the voltage and orientation criteria listed in Tables I and II.

The relative weight of positive criteria and standards for the upper and lower normal limits of the q, the R and the S wave voltages vary from one cardiologist to another. Generally accepted values are those from the tables of Ziegler's direct writer data (1951) or the photographic data of Liebman(1968), which is considered more accurately derived. The limits of normal can be taken at either two standard deviations (95 percentile) or three standard deviations (99 percentile) above or below the mean.

TABLE I

ECG CRITERIA FOR RIGHT VENTRICULAR HYPERTROPHY

Principal Criteria

I. Abnormally increased anterior forces as manifested by
   A) Increased R/S ratio in V1 or V2
   B) Increased terminal R or R' in V1 or V2
II. Abnormally increased rightward forces as manifested by
   A) Increased S wave in V5 or V6
   B) Decreased R/S ratio in V5 or V6
III. Right axis deviation of the mean cardiac vector

Minor Criteria

I. Abnormally deep q waves in V1
II. Upright T waves in V1 after 72 hours of age
III. QRS-T angle in excess of 60 degrees with T wave directed towards the left.

TABLE II

ECG CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY

Principal Criteria

I. Abnormally increased posterior forces as manifested by
   A) Abnormally deep S waves in V1 or V2
   B) R/S ratio less than lower limit of normal for age in V1 or V2
II. Abnormally increased leftward forces as manifested by
   A) Increased R wave in V5 or V6
   B) Increased R/S ratio in V5 or V6
III. Abnormally increased inferior forces as manifested by increased R wave in II or aVF.

Minor Criteria

I. Abnormally deep Q waves in V6 or II, III and aVF
II. Abnormally deep inverted T waves in left precordial leads in severe disease
III. QRS-T angle in excess of 60 degrees with T directed towards the right.
IV. Abnormally deep S wave in V5 or V6 with terminal SV2---representing an arbitrary definition of left posterobasal hypertrophy, which can occur in conjunction with a left ventricular pressure overload.

The empiric observation that increased voltages are correlated with ventricular hypertrophy has been reaffirmed so many times that electrocardiologists have concentrated more on explaining how the non voltage criteria---i.e. axis deviation of the mean cardiac vector, the septal vector, the terminal QRS vector, and the T wave ---provide clues to the diagnosis of ventricular hypertrophy.

AXIS DEVIATION OF THE MEAN CARDIAC VECTOR

There are no good reasons why the axis deviation and hypertrophy should be related. In fact, the two are usually considered separate entities. Grant (1953) has pointed out that left axis deviation neither supports nor rules out the diagnosis of left ventricular hypertrophy. With right ventricular hypertrophy, the issue is more controversial with electrocardiographers to support either position (Liebman, 1968).

SEPTAL VECTOR

Disturbances in the magnitude and duration of the initial septal vector have been an important ancillary point in the support of the diagnosis of ventricular hypertrophy. In right ventricular hypertrophy, for example, there may be an associated thickening of the right ventricular portion of the septum, which because of its increased size, causes a q in lead V1 (Liebman, 1968). A similar explanation may hold for the finding of a higher frequency of leftward and posterior initial forces in patients with right ventricular hypertrophy than in normal people (Fowler and Helm, 1953). In patients with cystic fibrosis and right ventricular hypertrophy, the initial vector was also on the average more leftward and

inferior than when there was no right ventricular hypertrophy.

Although Nadas(1972) has suggested that qR in V1 often on closer inspection turns out to be an rSR' pattern, which is more often the "rule rather than the exception in pediatrics," true leftward and posterior initial forces are considered abnormal. However, since a prolonged (more than .03 seconds) r in V1 can occasionally be found in right ventricular hypertrophy, qV1 is not always present in right ventricular hypertrophy. Nevertheless, the concept of septal hypertrophy associated with ventricular hypertrophy of the ipsilateral side is a good one and a prolonged , deep qV6 is also interpreted as good evidence for left septal hypertrophy.

TERMINAL QRS WAVE

Terminal QRS forces can also be confusing or helpful in determining the presence or absence of ventricular hypertrophy. In the normal heart, forces are directed posteriorly and somewhat leftward because the forces generated by the free wall of the left ventricle are considerably stronger and outlast those produced by the free wall of the right ventricle (Sodi-Pallares, 1970). When there is left ventricular hypertrophy, the leftward and posterior forces can be accentuated. However, when there is an accentuation of terminal posterior forces, particularly those directed rightward, whether this correlates with hypertrophy in the basal region of the left or the right ventricle is not easily determined. Furthermore, the presence of hypertrophy may or may not be associated with hemodynamic changes such as elevated pressure or volume flow in that ventricle.

Anatomically speaking, hypertrophy of the posterobasal region of the left ventricle is distinct from hypertrophy of the posterobasal region of the right ventricle or crista supraventricularis. The crista supraventricularis is a muscular ridge of tissue which separates the posterior portion of the outflow tract of the right ventricle from the inflow or sinus tract. In systole, it pulls the floor of the right ventricle toward the outflow tract(Kjellberg et al, 1954). According to Grant et al (1957) isolated crista supraventricularis hypertrophy is often seen in secundum atrial septal defects. On the other hand, left posterobasal hypertrophy has been associated with pressure overloads in the left ventricle (Liebman, 1968). Burch and DePasquale (1962) have pointed out that when there is occlusion or decreased blood flow through the left coronary artery, in particular, the left anterior descending branch, the result is loss of muscle in the anteroseptal and apical portions of the left ventricle. If the right corona ry artery is patent, then compensatory hypertrophy will take place in the basal portion of the ventricle to maintain left ventricular output.

In theory, hypertrophy of the posterobasal portion of the left ventricle and that of the right ventricle or the crista supraventricularis should be distinguishable electrocardiographically. The mean vector representing crista supraventricularis depolarization is oriented towards the right, superiorly and either anteriorly or posteriorly. In the standard leads I, II and III, there are deep, wide S waves or the deep S waves may be in leads I and II only. I n the precordial leads, there may be an rsR' with a relatively wide and slurred R' in Vl and deep, wide S waves are usually

present in V4, V5 and V6. On the other hand, in left posterobasal hypertrophy, the terminal QRS vectors are oriented rightwards, superiorly, and posteriorly and increased in both magnitude and direction.

Unfortunately, these electrocardiographic characterizations do not hold and a third entity, right bundle branch block, exists which can take on an essentially similar electrocardiographic picture making the diagnosis of posterobasal hypertrophy all the more uncertain. Metianu, Durand and Dauzier(1953), Liebman (1968), Kjellberg et al (1954) and others have pointed out how frequently diagnoses of right bundle branch block are made on the electrocardiogram of patients with coarctation of the aorta. This high frequency may or may not be an artifact, since the overlap between right bundle branch block, crista supraventricularis and left posterobasal hypertrophy on the ECG is great.

A number of suggestions have been made in order to improve the diagnostic potential of the terminal QRS forces. Among these is the finding by Blount et al (1957) that the duration of the initial r of the rsR' V1 pattern of crista supraventricularis hypertrophy is generally less than .025 seconds, while the rV1 of incomplete right bundle branch block has a duration greater than .03 seconds. Other suggestions involving the use of vector loops obtained with corrected orthogonal lead systems to examine the initial and terminal QRS deflections in right versus left ventricular posterobasal hypertrophy and right bundle branch block will be discussed in Section I. C.1.Theoretical Advantages of the Frank Corrected Orthogonal Lead System.

T WAVE

Sodi-Pallares' ideas on ventricular repolarization in hypertrophic ventricles, although unproven, provide insight on how aberrant T waves can serve as clues to the diagnosis of ventricular overloading. It is known that in the normal heart, depolarization of the free wall of the ventricles proceeds from subendocardium to subepicardium and that repolarization occurs from subepicardium to subendocardium. Sodi-Pallares (1970) explains this phenomenon by postulating that normal pressures within the ventricles causes relatively more subendocardial than subepicardial ischemia and that this causes the recovery process of the endocardium to be delayed longer than that of the subepicardium. Hence, positive T waves are seen in leads V5 and V6 of the normal electrocardiogram because the repolarization process of the dominant left ventricle can be represented by a dipole in which the negative pole precedes the positive pole and is moving away from the surface chest electrode. The deflection of repolarization is even more positive in magnitude with increases in intraventricular pressure because of the unmasking caused by lack of cancellation of the subendocardial electric forces by the epicardial forces of different direction or opposite polarity.

In contrast, according to Sodi-Pallares, in severe ventricular hypertrophy, the increased muscle thickness of the free wall of the ventricles delays the activation of the subepicardium and consequently its repolarization to a greater extent than the delay produced by the intraventricular pressure upon the subendocardium. Hence, the repolarization can be represented by a dipole in which the leading negative pole approaches closer to the electrode on the chest wall than the positive pole which follows. Consequently, one sees deeply inverted T waves over a ventricle which is severely hypertrophied.

I.B3. CORRELATION BETWEEN ECG CRITERIA AND HEMODYNAMIC FUNCTION

Ideally, the ability to predict hemodynamic values from electrocardiographic parameters should depend on the correctness of physiological and pathological insights, rather than on the degree of statistical correlation alone without adequate preprocessing of electrocardiographic and hemodynamic data. Unfortunately, an understanding of the physiologic bases for the magnitude and pattern of the measured body surface electric potentials is simply not available. Racial background, body size proportions, and other anthropometric differences which have been shown to account for differences in the electrocardiograms of patients with normal hearts (Walker, 1961; Traywick, 1973) have not been taken into account in most electrocardiographic studies. Hence, few of the statistical correlations which have been calculated can be of useful validity without an appreciation for all the factors which can influence the pattern on the electrocardiogram.

Despite the lack of full physiologic and pathological understanding, the predictive potential of electrocardiographic parameters have been studied by the computation of correlation coefficients. In the early days of electrocardiography, correlations involving the magnitude of the voltages and ventricular wall thickness, heart volume, and weights measured according to carefully described protocols were very popular. Yet, as limited as correlation studies in autopsy patients are in predicting antemortal hemodynamic function, "since often things are found (at autopsy) which may or may not have influenced the electrophysiologic phenomena before death (Kossman, 1970)," few studies of autopsy correlations have been done in patients of pediatric age. Scott wrote in

1960 that, to the best of his knowledge, there were only a few autopsy studies (Hollman, 1958) which examined the validity of ECG criteria for right ventricular hypertrophy but no extensive correlation studies for ECG criteria for left ventricular hypertrophy. Thus, one cannot logically expect to find a high correlation between electrocardiographic diagnoses or interpretations and autopsy findings in children.

In the 1950's, the ECG was studied as a predictor of hemodynamic function. Cayler et al (1958) described a correlation coefficient r of .78 between the height of the tallest r wave in the right precordial leads and the peak systolic intraventricular pressure in patients with pulmonic stenosis, but did not believe that this correlation was adequate to be of clinical usefulness. Other studies of the relationship between ECG parameters and the peak systolic intraventricular pressure and other guides to severity such as the valve pressure gradient in pulmonic stenosis have also revealed very low correlations (DePasquale and Burch, 1960; Penaloza et al, 1954). On the left side of the heart, Braunwald et al (1963) reported that there were no reliable correlations between the ECG and the severity of the obstruction in congenital aortic valvular stenosis. The term "left" or "right ventricular hypertrophy" in pediatric ECG terminology remains ambiguous, indicating only increased ECG electrocardiographic forces without any strong hemodynamic or anatomic correlation, such as increased peak systolic intraventricular pressure, increased volume overload, or increased autopsy weight of the heart.

I. C. THE FRANK SCALAR ELECTROCARDIOGRAM

At the Yale-New Haven Hospital, the XYZ electrocardiogram consists of three orthogonal leads simultaneously recorded at a rate of 200 mm/second. The top lead represents the x or right-left axis, the middle the y or superior-inferior axis and the bottom the z or anterior-posterior axis. In scanning the XYZ, one looks most importantly at the distribution of rightward and anterior forces as well as leftward and posterior forces which signify increased right and left ventricular forces, respectively.

The left and right maximal spatial voltages are calculated using the method popularized by Ellison & Restiaux (1972). The maximal spatial voltage is the magnitude of that instantaneous vector for which the value of the square root of $(x^2 + y^2 + z^2)$ is maximal. If a left maximal spatial voltage is specified, then x is necessarily positive or directed towards the left but can appear at any time in the QRS cycle. An analagous situation holds true for the right maximal spatial voltage. Each of these values is thought to bear a rough correspondence to the amount of ventricular mass and the degree of pressure and volume overloading of the ventricle on that particular (left or right) side.

XYZ displays of electrocardiographic information are intended to have normalized strength and anatomic orientation as well as theoretically limited departures from these ideals with changes in the position of the heart. The argument that corrected leads more accurately represent the electrical activity of the heart has been used to suggest that the Frank system

replace the standard twelve lead (ECG) system wherever electrocardiograms are obtained. However, in comparing the two systems from the standpoint of usefulness as a diagnostic tool, other equally important factors have to be considered. The advantages of a corrected lead system, including (1 a.) normalized strength and anatomic orientation, (1 b.) simultaneity of recording, (1 c.) adaptability to resolution or conversion into other lead systems and two dimensional loop displays and (2.) better correlations with hemodynamic parameters than have heretofore been obtained with the ECG, have to be weighed against (3.) the practical consideration of whether or not the clinician can gain as much information from the XYZ as the ECG. Each of these factors will be considered in the following discussion.

I.C.1. THEORETICAL ADVANTAGES OF THE FRANK CORRECTED ORTHOGONAL LEAD SYSTEM

A. Normalized Strength and Anatomic Orientation

The performance of a lead system is measured by the amount of variation in lead strength and lead direction when the heart or current source changes in location. Lead strength is defined as the ratio between the magnitude of the potential registered in a given lead and the strength of the current source giving rise to this potential. Lead direction is the direction of the axis upon which the cardiac vector is projected. Lead strength and lead direction in the ideally corrected lead system are arranged so that changes in location of the heart from individual to individual or with movement play no role in the genesis of the electrocardiographic tracing.

In practice, electrocardiographic lead systems are subject to enormous variation in lead strength and lead direction because the following four assumptions, necessary

to apply to most uncorrected lead systems, are not always justified:

1) The human body can be assumed to be spherical in shape.
2) The current source is located precisely in the center of the volume conductor.
3) The heart or physiologic current source is a point-like, fixed location single dipole.
4) The electrical conductivity of all body tissues is uniform.

In particular, unless all four assumptions are applicable to the standard twelve lead electrocardiogram (ECG), Einthoven's equilateral triangle hypothesis---i.e. that the sum of the voltages in the standard leads I, II and III of the frontal plane of the ECG is zero--- is false, thus invalidating the ECG determinations of axis deviation of the mean cardiac vector, voltage magnitude, and all other voltage and orientation parameters.

In the 1950's, Frank (Wolferth, 1953) calculated that the electric potentials found at the apices of an equilateral triangle on the surface of a sphere composed of a homogeneous conductor with a current dipole placed exactly at the center (i.e. satisfying all four assumptions for the non-corrected frontal plane lead system of Einthoven) would be in accord with Einthoven's equilateral triangle hypothesis. However, when the slightest eccentricity of the dipole was introduced into the calculations, the sum of the voltages in standard leads I, II and III was considerably different from zero.

Famous critics of Einthoven's equilateral triangle hypothesis included Burger and Milaan, who in 1946 published the famous Burger scalene triangle. In essence, the assymetrical lengths and angles of this triangle were designed to illustrate

the proper weighting and orientation of the voltages in standard leads I, II and III with respect to one another, if Einthoven's equilateral triangle hypothesis were to hold true. The bulk of scientific information has supported the contention that the voltages of the standard leads, when scaled and oriented according to the proportions and angles of Burger's triangle, would provide a more accurate calculation of the axis and magnitude of the mean cardiac vector.

The Frank system is a corrected lead system which was developed by using realistic models of cardiac electrical activity which do not rely as heavily upon the first two of the four assumptions mentioned above for their accuracy. In particular, in designing the Frank lead system, a homogeneous torso rather than a sphere was constructed to simulate the volume conductor in order to minimize the errors inherent in the first assumption (of a spherically shaped volume conductor). In addition, in designing the Frank system, voltages were measured throughout the surface of the chest while the current source (a single dipole) was placed in different parts of the torso. By adjusting lead strengths with resistances which were empirically derived to account for the geometrical relationship between the cardiac generators and the body surface, the effective electrical lead axes were made to coincide as closely as possible with known (calculated) potentials along the anatomic orthogonal axes. A series of maneuvers was also elaborated in the original paper (Frank, 1956) to place the electrodes of the Frank system around the effective electrical center of ventricular depolarization. These efforts were undertaken to minimize the errors inherent in the second assumption, that the current source was a fixed point in the center of the thoracic cavity.

While the first and second assumptions are thus no longer necessary for the accuracy of the Frank lead system, neither the third assumption that a single dipole source is the most accurate model of electrical activity in the myocardium nor the fourth assumption that the electrical conductivity of all body tissues is uniform are justified.

It cannot be known with exact certainty what percent of the true electrical activity of the heart can be represented by a single dipole model, since it is mathematically impossible to calculate the distribution of a set of electrical charges from the surface potential information alone. Thus, while Schmitt (1953) and Frank (1955) estimate that in normal subjects body surface potentials have a ninety percent or more content attributable to the fixed location single dipole, Barnard, Sallin and Holt (1970) found that the single dipole model accounted for only sixty percent of the body surface potential.

The non-dipolar contribution can be from either higher singularity sources or the movement of a single dipole. If it is the former, then the designers of the Frank system have committed a grave error, since the vector sum over all the dipole sources may vanish at a moment when the quadrupole or a higher order term determines a significant non- zero electrical potential. On the other hand, if the non-dipolar contribution results from movement of a single dipole, then the Frank lead system no longer provides this information since the effect of changes in location or movement of the dipole were suppressed in its design. In this case, the ECG would provide additional information since it was not designed to suppress information from the movement of a dipole.

Although Pipberger wrote in 1959 that inhomogeneities in electrical resistivity did not play a major role in lead performance, there have been frequent subsequent reports of new data showing the contrary. Since all of the presently used orthogonal lead systems including the Frank were based upon experiments using homogeneous torsos, there is no logical basis to assume that any of them should be completely accurate in heterogeneous man. Grayzel and Lizzi (1969) have published data showing that there is considerable variation in the magnitudes and angles of vectorcardiograms obtained by each of six major vectorcardiographic systems in popular use (including the Frank corrected orthogonal lead system) on heterogeneous torsos in which the varying resistances of lung, heart blood, and chest wall were simulated. The large variation found in these six lead systems, including three corrected systems, seriously questions the lead performance of all vectorcardiographic systems in current use.

For the clinician, the major difference between ECG and XYZ tracings is usually in the voltage obtained along the Z axis or, in ECG terminology, with the unipolar precordial leads. According to Guntheroth (1972), the correlations between the X lead and lead I and between the Y lead and lead aVF are good, but there are often large discrepancies between V2 and Z, especially in situations with right axis deviation. Commonly, as Guntheroth pointed out, an rS will be seen in V2 suggesting posterior forces while the Z lead will show balanced anterior-posterior forces. Generally, the conclusion is that the S in V2 actually reflected rightward forces, although, given errors in the dipole and homogeneity assumptions of the Frank scalar XYZ system, it is quite possible that instead the XYZ system has suppressed true posterior forces.

In fact, insofar as the chief difference between the XYZ and the ECG lies in the z or anterior-posterior leads, it is important to realize that the differences are not simply the unwitting example of differences between a corrected and an uncorrected lead but a reflection of two entirely different electrocardiographic approaches: the semi-direct approach versus the correction for eccentricity approach. Whereas the corrected orthogonal lead systems are intended to minimize the effects of an eccentric position of the current source, the unipolar precordial leads of the ECG were devised on the theoretical premise that primarily the myocardium directly underneath the exploring electrode was being recorded. This theory, called the semidirect theory of Wilson, had its origins in the work of Lewis, who in the years 1910-1916 realized that QRS deflections were large and easily recognized when one electrode (the exploring electrode) was placed directly on the epicardial surface of the heart while a second electrode (the indifferent electrode) was essentially grounded on the chest wall. Wilson's modification and extension of this theory was to develop a central terminal which connected and grounded all electrodes to the body save the exploring electrode. The resultant exploring electrode was to become the unipolar lead used in the ECG today.

I.C.1. B. SIMULTANEITY OF RECORDED LEADS

The recording of simultaneous leads makes it possible to more accurately localize the origin and direction of forces on an electrocardiogram. In a patient with an arrhythmia or otherwise rapidly changing ECG pattern, interpretation based on measurements taken on sequential leads is not as accurate as when

there is a stable state in which sequential beats are considered to be comparable. Hence, the simultaneity of recorded leads in the XYZ system, which also saves time and paper, has definite advantages over the ECG.

I. C. 1. C. RESOLUTION INTO OTHER LEAD SYSTEMS

Despite the contemporary interest in developing corrected, orthogonal lead systems, electrocardiograms taken with corrected, orthogonal leads may not necessarily be the ones which will give the most sensitive and specific separation between normal hearts and those with pressure or volume overloading. As Gamboa, Hugenholtz and Nadas (1966) pointed out, uncorrected cube systems may be advantageous in the study of certain time vectors due to artifacts in their lead design. The artifacts of the ECG, for example, may account for its being the most popularly used electrocardiographic tool for the diagnosis of ventricular hypertrophy.

The potential for mixing or resolving ideally corrected electric activity into leads in any direction to maximize a separation of electrocardiograms into those of patients with and those of patients without a particular electrocardiographic feature is clearly one advantage of the corrected lead system. The classic example of resolution of the orthogonal lead systems into different displays is the two dimensional planar loop or vectorcardiogram. Additional diagnostic information which is not easily found in the examination of scalar orthogonal leads are thought to be derived from examination of the vector loops. For example, increased initial leftward and posterior forces have been found in the vectorcardiogram loops of patients with left ventricular pressure overloads as opposed to the more rightward and anterior forces of patients with normal hearts or left ventricular volume overload (Hugenholtz and Gamboa, 1964; Krovetz

et al, 1969; Ellison and Restiaux, 1972).

Elliot et al (1965) have shown that posteriorly directed forces in ECG's can be misleading in terms of indicating whether it is the left or the right ventricle which is hypertrophied. However, they suggest that if a vectorcardiogram is taken, posteriorly and rightwardly directed forces indicate right ventricular overload while posteriorly and leftward directed forces indicate a left ventricular overload. Batchlor (1970) has also done pioneering studies to show how non-orthogonal resolutions of corrected orthogonal leads can provide new axes with maximum potential for distinguishing right ventricular overload and other cardiac disease.

I. C. 2. XYZ ELECTROCARDIOGRAPHIC PARAMETERS AND THEIR POTENTIAL IN EVALUATING HEMODYNAMIC FUNCTION

The diagnostic potentials of the XYZ, like the ECG, are limited by the inadequacy of statistical correlations as well as inadequate understanding of the physiologic bases for the magnitude and shape of the body surface electrocardiographic potentials. In a paper entitled "The Love at First Sight Effect in Research," Pipberger (1968) provided some useful insight into the value of the correlations which have been published and publicized in orthogonal electrocardiography. Taking as an example his own studies on the correlation between age and the maximal spatial voltage of the QRS, Pipberger drew a plot showing how the correlation coefficient between these two variables changed as the number of patients studied increased. With twenty-five patients, the correlation coefficient was 0.6. With fifty patients, it was 0. With one hundred forty patients, it was -0.4. And with five hundred patients, r settled down to about -0.3.

Pipberger concluded that at least two hundred patients are needed for a good correlation between the normal electrocardiogram and other hemodynamic parameters. Even more patients are necessary to demonstrate a valid correlation coefficient between an abnormal electrocardiographic finding and a hemodynamic parameter. Yet, the average number of patients studied in most papers which have calculated correlation coefficients is sixty-five.

In addition, various sophisticated statistical techniques have been devised for the analysis of electrocardiographic data. One example is a technique for the calculation of the standard deviation of a set of angular data (Liebman et al, 1966). It is not clear however whether this technique is used in even some of the best known monographs in orthogonal electrocardiography, such as the one by Ellison and Restiaux (1972).

A fair assessment of the statistical correlations in the literature must take into account the difficulty of accumulating large numbers of patients with cardiac defects and the questionable wisdom of applying mathematical rigor to the imprecise science of electrocardiographic interpretation. However, whether or not statistically significant, the calculated correlations are valuable for providing insight into the expected patterns of correlations.

Generally, it has been felt that the pressure overloaded situations provide better correlations between the maximal spatial voltage magnitude and the peak systolic intraventricular pressures (Hugenholtz and Gamboa, 1964; Ellison and Restiaux, 1972). This was related by Hugenholtz and Gamboa (1964)

to the more important role of pressure as opposed to volume overloads in augmenting myocardial oxygen consumption, which constitutes an important stimulus for cardiac hypertrophy. Furthermore, the well known reports of Hugenholtz and Gamboa and Ellison and Restiaux (1972) seem to indicate that the right and the left peak systolic intraventricular pressures have comparable correlation coefficients with the magnitude of the right and left maximal spatial voltages, respectively. An r value of 0.85 was calculated by Hugenholtz and Gamboa (1964) in fifty patients for the right ventricle and 0.78 was calculated for the left ventricle using the Frank system. Ellison and Restiaux (1972) found the values 0.73 and 0.82 for the right and left sides of the heart, respectively, in one hundred patients.

Calculations of correlation coefficients relating maximal spatial voltages and hemodynamic parameters in volume overloaded ventricles are not easily interpreted. Logically, if the degree of cardiac hypertrophy is correlated with the magnitude of the electrocardiographic voltages, the stimulus to the cardiac hypertrophy---be it a pressure or volume overload---is best correlated with the magnitude of the electrocardiographic voltages. One expects to find in a volume overloaded situation a much greater correlation between the maximal spatial voltage and some hemodynamic parameter quantifying the magnitude of the cardiac shunt or volume overload than between the maximal spatial voltage and peak systolic intraventricular pressure. Yet, Ellison and Restiaux(1972) found comparable correlation coefficients between the right maximal spatial voltage and Qp/Qs (0.70) and between the right maximal spatial voltage and right

ventricular peak systolic pressure (0.81) in forty patients over the age of two years with the diagnosis of atrial septal defect. Whether this result is due to the small size of the series or physiologic principles which are not well understood is not clear.

Volume overloaded lesions involving valvular insufficiency or shunts without a distal mixing chamber are difficult to quantitate mathematically. Hence, in these lesions, it is hard to determine the degree of correlation between electrocardiographic voltages and the severity of these lesions because an accurately calculated numerical parameter quantifying the volume overload is not available. Nevertheless, empirically, it has been observed that it is more likely for a left ventricular volume overload to give an electrocardiographic picture similar to a left ventricular pressure overload than for a right ventricular volume overload to show increased right ventricular forces electrocardiographically.

Other parameters which can be calculated from an XYZ electrocardiogram, such as the time of occurrence of a maximal spatial vector, its azimuth (angle between X axis and vector in the XZ plane) and elevation (angle between the X axis and vector in the XY plane), and similar information on the 10 millisecond, 20 millisecond, P and T wave vectors have been collected in various previous studies (Aziz et al, 1971; Ellison and Restiaux, 1972 (reference 48)). But aside from the possible association between increased initial leftward and posterior forces and a left ventricular pressure

increase, the diagnostic potential of these other parameters remains largely speculative.

In the pediatric age group, only Blumenschein et al (1972) have tested whether higher correlations between electrocardiographic parameters in corrected lead systems and hemodynamic parameters might be used to develop XYZ criteria for the diagnosis of cardiac defects in an unknown population of pediatric age. In their study, all of ten patients with atrial septal defects were correctly diagnosed by the presence of Sx, Ry and Sz values whose vector magnitude fell outside of the normal range.

I. C. 3. DISADVANTAGES OF THE XYZ

The critical factor in deciding whether one will use the XYZ or the ECG in clinical practice is the relative amount of information which each lead system provides about cardiac function. Previous workers have compared the electrocardiographic content of ECG's with the content of three lead orthogonal electrocardiograms taken on adult patients and concluded that the amount of clinical information that can be obtained from the ECG is greater not only owing to increased physician familiarity but to the larger number of leads displayed in the ECG. Abildskov (1958) studied 71 ECG's and found that in 9.8 % of the cases, clinically significant abnormalities contained in the standard ECG could not be recovered from the orthogonal leads. Pipberger studied 261 patients in 1961 and found that in 7.3% of the orthogonal electrocardiograms, clinical information found in the ECG could not be located. However, when the orthogonal leads were resolved to form a rough approximation of the standard twelve lead electrocardiogram, there was only one case in the 261 in which a questionable discrepancy existed between the ECG and XYZ.

In addition, as Von der Broeben (1965) has pointed out, besides taking additional time, the summation of three bits of information, the x, the y and the z components of the vector representing electrical depolarization, in the calculation of the maximal spatial voltage involves a loss of information which may be vital to the optimal separation of normal from abnormal electrocardiograms. The three dimensional space defined by the maximal spatial voltage encloses decision regions which could

be separated by better fitting envelopes and constitutes an unreliable definition of the normal ventricular depolarization.

II. METHODS & MATERIALS

A. The Clinical Study

The data presented in this study include standard twelve lead electrocardiograms (ECG's) and Frank scalar XYZ's on seventy-four patients varying in age from 8 months to 17 3/12 years, all of whom underwent cardiac catheterization. Data were chosen for presentation because they represent situations of definite or possible right ventricular pressure overload (isolated pulmonic stenosis, Tetralogy of Fallot, ventricular septal defect with pulmonary artery band), left ventricular pressure overload (aortic stenosis, coarctation of the aorta), left ventricular volume overload (patent ductus arteriosus, ventricular septal defect) or right ventricular volume overload (atrial septal defect). In addition, for the diagnoses of isolated pulmonic stenosis, aortic stenosis, coarctation of the aorta and patent ductus arteriosus, all cases with photographic XYZ's available (taken on all catheterization patients at Yale-New Haven Hospital from mid-1972 to early 1974) were included.

ECG's were recorded on a conventional Hewlett Packard ECG unit. XYZ drawings were recorded with a Schwarzer electrocardiograph using Frank lead electrodes placed in the standard positions along the fourth intercostal space ("nipple line") or lower: lead A was placed in the left mid axilla, E in the middle of the sternum, C midway between these two, I in the right mid axilla, and lead M in the midback.

Standard calibration was 10mm equals 1 mv in almost all instances. Paper speed was 250 mm/sec on the photographic XYZ's taken on the Hewlett Packard Sanborn 1507-11A lead

programmer and 200 mm/sec on the direct writing Schwarzer Frank scalar machine.

A mm ruler was used for measurements from the top of the isoelectric line to the peak of the positive deflection and the measurements from the bottom of the isoelectric line to the most negative point of the downward deflection. For the XYZ, simultaneous X, Y and Z amplitudes were measured at intervals of .005 seconds or closer, using a Wang 600 computer to choose the maximal spatial vector, as defined by Ellison and Restiaux (1972). Azimuth and elevation were also calculated by the Wang computer using the measured amplitudes. In addition, spatial vector magnitudes, azimuth and elevation were calculated for the first .01 and .02 seconds on all direct writer XYZ's.

Catheterization was carried out using standard techniques and equipment. Right ventricular systolic pressures less than 30 mm Hg were considered normal. Upper limits of normal for left ventricular systolic pressures were taken at two standard deviations in infants from the data of Moss and Adams (1962) and at the 90th percentile for older children from data collected by Conde (1966).

B. Criteria

In this study, one compares the XYZ and ECG findings of patients who subsequently underwent cardiac catheterization on the pediatric cardiology service during the first six months of 1974. All XYZ's and ECG's were interpreted according to the criteria outlined below.

Three sets of XYZ criteria were designed to separate normal patients from those with abnormally increased ventricular forces. "Abnormally increased XYZ forces" is defined here to imply values in excess of one standard deviation above the mean values given in the Appendix, Table III, of the

text by Ellison and Restiaux (1972).

The first set of criteria identified patients with abnormally increased ventricular forces in either the rightward or leftward direction, regardless of anterior=posterior orientation.

The second set of criteria identified patients with abnormally increased rightward and anterior forces or those with abnormally increased leftward and posterior forces from those who had forces which were either normal or abnormally increased in magnitude but did not have the orientations specified above.

The third set of criteria identified patients with abnormally increased posteriorly directed forces, regardless of their left-right orientation.

The three sets of XYZ Criteria for increased ventricular forces are specified in Table III (next page).

ECG Criteria for right and left ventricular hypertrophy used in this study were those shown in Tables IV and V. "Abnormally increased" as used in these two tables is defined as exceeding two standard deviations above the mean, according to age, as given in the standard tables of Liebman (1968).

TABLE III

XYZ CRITERIA FOR INCREASED VENTRICULAR FORCES

First Set: XYZ VOLTAGE Criteria for Increased Ventricular Forces

A. Increased right ventricular forces are present if the right maximal spatial vector (RMSV) voltage exceeds one standard deviation above the mean, according to age, as specified in the Appendix, Table III, of the text by Ellison and Restiaux(1972).

B. Increased left ventricular forces are present if the left maximal spatial vector (LMSV) voltage exceeds one standard deviation above the mean, according to age.

Second Set: XYZ VOLTAGE & ORIENTATION Criteria for Increased Ventricular Forces

A. Increased right ventricular forces are present if the right maximal spatial vector voltage exceeds one standard deviation above the mean, according to age, and is oriented anteriorly.

B. Increased left ventricular forces are present if the left maximal spatial vector voltage exceeds one standard deviation above the mean, according to age, and is oriented posteriorly.

Third Set: POSTERIOR ORIENTATION Criteria for Increased Left Ventricular Pressure

Left ventricular pressure overload is present if the voltage of either maximal spatial vector, left or right, exceeds one standard deviation above its mean value, as given according to age by Ellison and Restiaux (1972) and the vector is oriented posteriorly.

TABLE IV

ECG CRITERIA FOR RIGHT VENTRICULAR HYPERTROPHY

Major Criteria

I. Abnormally increased anterior forces as represented by abnormally increased R/S ratio in Vl. (An "abnormally increased" ratio is defined here as one which exceeds the upper limits of normal, according to age, as given in Table 2 of the text by Guntheroth (1965).

II. Abnormally increased rightward forces as represented by abnormally increased SV6, according to age, using as the upper limit of normal, two standard deviations above the mean in the standard tables of Liebman (1968).

III. Right axis deviation of the mean cardiac vector as represented by a mean cardiac vector whose axis lies outside the normal range as given according to age in Figure 13 of the text by Guntheroth (1965).

Minor Criteria

I. Abnormally deep q waves in Vl (deeper than the maximal upper limit as given according to age in Table 17 of the text by Ziegler (1951).

II. Upright T waves in Vl after 72 hours of age.

III. QRS-T angle in excess of 60 degrees with T directed towards the left.

IV. Any of the Principal Criteria described in Table I which is not being used as a Major Criteria can constitute a minor criterion.

The diagnostic interpretation of DEFINITE right ventricular hypertrophy was made when all three of the major criteria were positively met.

PROBABLE right ventricular hypertrophy when two of the major criteria and a minor criterion from a category with a different Roman numeral were met.

POSSIBLE right ventricular hypertrophy if one of the major criteria other than right axis deviation and one minor criterion

from a category with a different Roman numeral were met. Alternatively, two minor criteria from categories with different Roman numerals would also, if positive, suggest POSSIBLE right ventricular hypertrophy.

TABLE V

ECG CRITERIA FOR LEFT VENTRICULAR HYPERTROPHY

Major Criteria

I. Abnormally increased posterior forces represented by abnormally deep S wave in V1. (An "abnormally increased" or deep wave is one which exceeds two standard deviations above the mean, according to age, as given in the tables of Liebman (1968).

II. Abnormally increased leftward forces as represented by abnormally increased R wave in V6.

III. Abnormally increased inferior forces as represented by abnormally increased R wave in lead II or aVF.

Minor Criteria

I. Deep q waves in V6 or leads II, III and aVF (exceed the maximum upper limit in Table 16 or Table 17 of the text by Ziegler (1951)).

II. Deep inverted T waves in left precordial leads (exceed maximum upper limit of normal in Table 32 of the text by Ziegler (1951)).

III. QRS-T angle in excess of 60 degrees with T directed towards the right.

IV. Any of the Principal Criteria described in Table II which is not being used as a Major Criteria can constitute a minor criterion.

The diagnosis of DEFINITE left ventricular hypertrophy was made when at least two of the major criteria and one minor criterion were positively met.

PROBABLE left ventricular hypertrophy when one major criteria and at least three minor criteria each from categories with a different Roman numeral were met.

POSSIBLE left ventricular hypertrophy if at least two minor criteria from categories with different Roman numerals were met.

Right Ventricular Conduction Delay (RVCD) was arbitrarily defined by the presence of (1) slurred, elevated SV5 or S V6; (2) terminal anterior (R) forces in V2 ; and (3) a QRS interval prolonged above the upper limits of normal (Guntheroth, 1965) but less than .10 seconds in duration.

Left Posterobasal Hypertrophy (PBH) was arbitrarily defined by the presence of (1) an abnormally increased S V5 or S V6 (exceed more than two standard deviations above the mean for given age, according to Liebman (1968) tables; (2) terminal posterior (S) forces in V2, regardless of magnitude; and (3), if right ventricular hypertrophy is present, this diagnosis is suppressed.

Biventricular Hypertrophy (BVH) was present if there was at least POSSIBLE right ventricular hypertrophy and POSSIBLE left ventricular hypertrophy; or, the sum of R and S in the midprecordial leads V2, V3, V4 or V5 were abnormally elevated (exceeded more than the 95th percentile or two standard deviations above mean, according to the tables of Liebman (1968)).

III. RESULTS

The seventy-four patients for whom data is presented in this study had the following lesions, which can be grouped according to their potential hemodynamic effect. Each group will be discussed in the order shown.

LESION	NUMBER OF PATIENTS
RIGHT VENTRICULAR PRESSURE OVERLOAD	
Pulmonic Stenosis	13
Ventricular Septal Defect with Pulm. A. Band	4
Tetralogy of Fallot	6
Ventricular Septal Defect	7
Atrial Septal Defect	5
LEFT VENTRICULAR PRESSURE OVERLOAD	
Aortic Stenosis	12
Coarctation of the Aorta	9
LEFT VENTRICULAR VOLUME OVERLOAD	
Patent Ductus Arteriosus	18
Ventricular Septal Defect	7
RIGHT VENTRICULAR VOLUME OVERLOAD	
Atrial Septal Defect	5

RIGHT VENTRICULAR PRESSURE OVERLOAD
PATIENTS WITH PULMONIC STENOSIS

A diagnosis of isolated pulmonic stenosis was found at catheterization in thirteen patients, ranging in age from one to thirteen years. With one exception, all patients had an elevated peak systolic right ventricular pressure (i.e. 30 or more mm Hg). There were no false positives and, hence, a specificity (specificity = 1- percentage of false positives) of 1 using either the ECG, the XYZ voltage, or the XYZ voltage and orientation criteria. The percentages of false negatives or patients with elevated peak systolic right ventricular pressures (RVP) not identified by each set of criteria were 1/12 using the ECG criteria, 5/12 using the XYZ voltage criteria, and 12/12 using the XYZ voltage and orientation criteria.

In other words, the sensitivity (1 - percentage of false negatives) of the ECG criteria (11/12) was highest without sacrificing specificity. The low sensitivity of the XYZ voltage criteria (7/12) can be explained by the presence of increased right maximal spatial vector (RMSV) voltage in only seven of the twelve patients with elevated RVP. The posterior orientation of the RMSV in eleven of the twelve patients accounts for the even worse performance of the XYZ voltage and orientation criteria. How these twelve patients could have had elevated right ventricular pressures without increased rightward and anterior forces will be discussed in a later section.

Of the thirteen patients, elevated right ventricular pressure was correctly identified by both ECG and XYZ voltage criteria in eight cases, incorrectly evaluated (false negative) by both lead system criteria in one case (J. B.), and the ECG was correct while the XYZ was incorrect in four instances (L. W., C. P., W. C., and V. P.).

In general, the magnitude of the maximal spatial voltage followed the increase in systolic right ventricular pressure and there was a correlation coefficient of 0. 61 (p less 0. 05). The regression equation for this relationship was

RVP = 17 + 43 (RMSV) $\pm$ 26 mm Hg.

TABLE VI A

PATIENTS WITH PULMONIC STENOSIS

PATIENT	AGE	RVP	PULM. A. PRESSURE	RMSV & ORIENTATION	ECG DIAGNOSIS
J. F.	5 8/12	25/5	20	1. 34 high nl P	NO RVH
V. P.	10 1/12	37/7	15/8	1. 23 high nl A	POSS RVH
J. B.	6 6/12	40/5	16/10	1. 07 normal P	NO RVH
L. W.	12 8/12	47/7	14/5	. 83 low nl P	POSS RVH
W. C.	12 10/12	65/10	20/10	. 58 decr P	POSS RVH
K. E	5 10/12	70/47	15/8	1. 90 incr P	DEF RVH
C. P.	5 1/12	80/10	15/10	1. 14 normal P	PROB RVH
H. F.	4 11/12	100/7	14/6	1. 94 incr P	DEF RVH
M. T.	9 8/12	90/10	16/9	1. 33 incr P	PROB RVH
L. F.	4 10/12	100/11	22/18	2. 12 incr P	DEF RVH
D. C.	11 9/12	105/9	20/7	1. 91 incr P	DEF RVH
J. C.	1	110/		1. 97 incr P	DEF RVH
L. L.	13 3/12	130/9	20/8	1. 44 incr P	DEF RVH

TABLE VI B

RESULTS IN PATIENTS WITH ISOLATED PULMONIC STENOSIS
ACCURACY OF ECG AND XYZ IN IDENTIFYING
PATIENTS WITH ELEVATED RIGHT VENTRICULAR PRESSURES

Criteria	False Positive	Specificity (1 - f p)	False Negative	Sensitivity (1 - f neg)
ECG	0/1	1/1	1/12	11/12
XYZ VOLTAGE	0/1	1/1	5/12	7/12
XYZ VOLT. & ORIENTATION	0/1	1/1	12/12	0/12

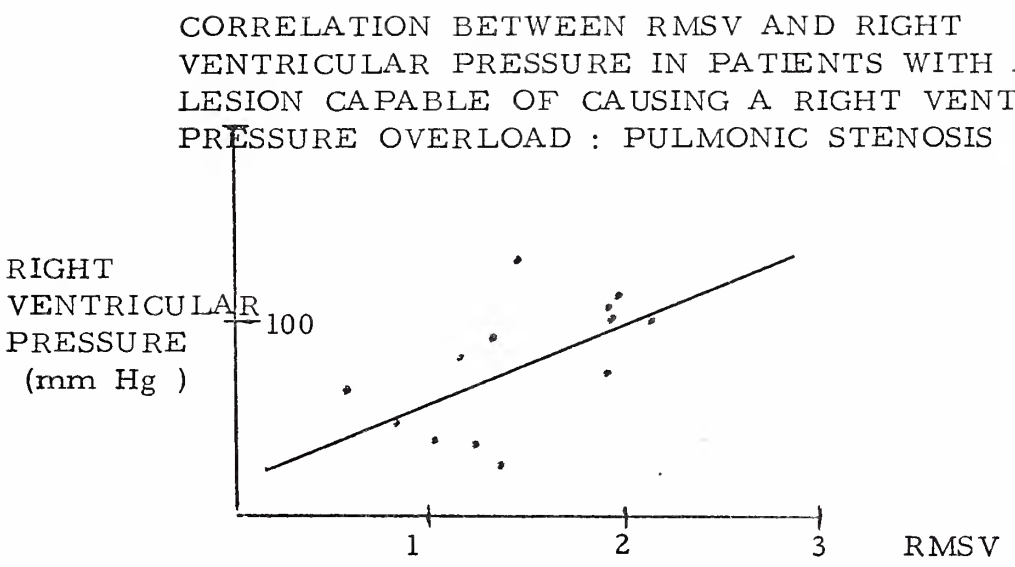

Figure 1. RMSV versus right ventricular pressure in thirteen patients with pulmonic stenosis. Correlation coefficient r = 0.61 (p less .05). Regression equation for relationship between RMSV and right ventricular pressure is RVP=17.3 +42.7(RMSV) $\pm$26 mm Hg.

PATIENTS WITH VENTRICULAR SEPTAL DEFECT AND PA BAND

There were four patients with a ventricular septal defect and pulmonary artery band (ages ranging from 2 10/12 to 5 9/12) with a lowest right ventricular systolic pressure of 90 to a highest of 140. Since there were no patients with normal right ventricular pressure, the specificity of the various criteria cannot be compared. However, the sensitivity of the criteria for RVH in identifying elevated right ventricular pressures were, in descending capability, ECG 4/4, XYZ voltage criteria 2/4, and XYZ voltage and orientation criteria 1/4.

In the two instances in which the ECG criteria were correct but the XYZ voltages misleading, the RMSV were either normal or low normal in magnitude. Three of the four RMSV were oriented posteriorly and one anteriorly---the former three being inappropriately oriented if one believes that increased anterior and rightward forces should accompany elevations in right ventricular pressure.

There was a high inverse correlation between the magnitude of the right ventricular pressure and the RMSV, r being -0.87 (p more than .1). The regression equation for this relationship was RVP (mm Hg) = 150 + (-23) (RMSV) $\pm$ 9.8 .

TABLE VII A

PATIENTS WITH VSD AND PA BAND

PATIENT	AGE	RVP	PA P	RMSV	ECG DIAGNOSIS
J.Z.	2 10/12	90/8	20/8	1.93 incr	DEF RVH
T.T.	5 9/12	95/10	95/10	2.80 incr	DEF RVH
M.M.	3 8/12	120/7	27/18	1.30 nl	POSS RVH
J.C.	2 11/12	140/13	110/40	.75 low nl	DEF RVH

TABLE VII B

RESULTS IN PATIENTS WITH VSD AND PA BAND
ACCURACY OF ECG AND XYZ CRITERIA IN IDENTIFYING
PATIENTS WITH ELEVATED RIGHT VENTRICULAR PRESSURES

Criteria	False Positive	Specificity	False Negative	Sensitivity
ECG	0/0	1 - fp	0/4	4/4
XYZ VOLTAGE	0/0	1 - fp	2/4	2/4
XYZ VOLT. & ORIENTATION	0/0	1 - fp	3/4	1/4

PATIENTS WITH TETRALOGY OF FALLOT

A diagnosis of Tetralogy of Fallot was found at catheterization in six patients, ranging in age from 8/12 to 6 6/12 years of age. The fact that all had elevated right ventricular pressures was correctly identified by the ECG and the XYZ voltage criteria. When the XYZ voltage and orientation criteria were used, there was a false positive rate of 3/6 and a sensitivity of 3/6. The absence of anterior orientation of the RMSV in the three inaccurately diagnosed patients accounted for the low sensitivity of the XYZ voltage and orientation criteria.

While all the RMSV's and RVP's were elevated, the correlation coefficient calculated between these two variables was not high. With $r = -0.56$ ($p > .1$), the regression equation was RVP = 117 + (-7.2) (RMSV) $\pm$ 3.2 mm Hg.

TABLE VIII A

PATIENTS WITH TETRALOGY OF FALLOT

PATIENT	AGE	RVP	RMSV	ECG DIAGNOSIS
Jm. G.	5 9/12	100/4	2.01 incr	DEF RVH
M.E.	2 5/12	100/5	2.08 incr	DEF RVH
L. D.	6 6/12	100/6	1.80 incr	DEF RVH
J.Q.	3 7/12	105/5	2.05 incr	DEF RVH
M.Z.	1 2/12	106/10	2.16 incr	DEF RVH
Jr. G.	8/12	110/6	1.28 incr	DEF RVH

TABLE VIII B

RESULTS IN PATIENTS WITH TETRALOGY OF FALLOT
ACCURACY OF ECG AND XYZ CRITERIA IN
IDENTIFYING PATIENTS WITH ELEVATED RIGHT
VENTRICULAR PRESSURES

Criteria	False Positive	Specificity	False Negative	Sensitivity
ECG	0/0	1 - fp	0/6	6/6
XYZ VOLTAGE	0/0	1 - fp	0/6	6/6
XYZ VOLT. & ORIENTATION	0/0	1 - fp	3/6	3/6

PATIENTS WITH ISOLATED VENTRICULAR SEPTAL DEFECT

The lesion of isolated ventricular septal defect was found at catheterization in seven patients ranging in age from 3 11/12 to 12 7/12 years of age and with right ventricular pressures ranging from a low of 22 mm Hg to a high of 120 mm Hg. Application of the ECG criteria to the one patient who had a normal right ventricular pressure (i.e. less than 30 mm Hg) resulted in a false positive diagnosis and an ECG specificity of 0/1. In contrast, both the XYZ voltage and the XYZ voltage and orientation criteria had a specificity of 1/1 and no false positives.

In patients with elevated right ventricular pressure, the ECG had no false positives and a sensitivity of 6/6. In the same group of patients, the XYZ voltage criteria had a false negative rate of 3/6 and therefore sensitivity of 3/6 while the XYZ voltage and orientation criteria had a false negative rate of 6/6 and 0 sensitivity. The posterior orientation of the right maximal spatial vectors accounted for the poor performance of the XYZ voltage and orientation criteria.

The presence of anatomic biventricular hypertrophy in a ventricular septal defect can lead to cancellation of opposing electric forces and account for the absence of increased maximal spatial voltages. The correlation coefficient calculated between the right ventricular pressure and the RMSV was 0.14 (p more .1). The corresponding regression equation was RVP = 53.3 + 8.53 (RMSV) $\pm$ 31.4 mm Hg.

TABLE IX A

PATIENTS WITH VENTRICULAR SEPTAL DEFECT

PATIENT	AGE	RVP	PA P	RMSV & ORIENTATION	ECG DIAGNOSIS
B.M.	7 4/12	22/2	22/10	1.27 high nl post.	PROB RVH
R.R.	3 11/12	36/3	34/12	1.62 incr post.	POSS RVH
B. Mo.	10 7/12	40/11	30/15	1.24 incr post.	NO RVH
K.S.	4 6/12	75/8	75/30	1.28 normal post.	DEF RVH (BVH)
J.A.	11/12	80/2	80/30	.55 decr post	POSS RVH
L.M.	10/12	85/6	85/25	2.45 incr post.	DEF RVH (BVH)
R.C.	12 7/12	120/10	120/60	1.52 high nl ant.	PROB RVH (BVH)

TABLE IX B

RESULTS IN PATIENTS WITH VENTRICULAR SEPTAL DEFECT
ACCURACY OF ECG AND XYZ CRITERIA IN
IDENTIFYING PATIENTS WITH ELEVATED RIGHT
VENTRICULAR PRESSURES

Criteria	False Positive	Specificity	False Negative	Sensitivity
ECG	1/1	0/1	0/6	6/6
XYZ VOLTAGE	0/1	1/1	3/6	3/6
XYZ VOLT. & ORIENTATION	0/1	1/1	6/6	0/6

PATIENTS WITH ATRIAL SEPTAL DEFECT

The diagnosis of isolated atrial septal defect was found at catheterization in five patients ranging in age from 11/12 to 8 7/12 years of age and with right ventricular pressures ranging from 20/2 to 37/6 mm Hg. In the three patients in whom right ventricular systolic pressure was less than 30 mm Hg and considered normal, the ECG diagnosed two as RVH. Hence, the ECG criteria had a false positive rate of 2/3 and sensitivity of 1/3, while only one of the three met criteria for elevated right ventricular pressure according to the XYZ voltage or the XYZ voltage and orientation criteria. Therefore, in this instance, the XYZ criteria proved more specific.

In the two patients who did have elevated right ventricular systolic pressures, both were correctly diagnosed with the ECG RVH criteria (i.e. ECG false negative rate of 0 and sensitivity 2/2). Neither were correctly identified using the XYZ voltage or the XYZ voltage and orientation criteria. False negative rates and sensitivity for these two latter criteria were 2/2 and 0/2, respectively. The correlation coefficient calculated for the RVP and RMSV in atrial septal defect was $r = -0.475$ (p more .1). The regression equation for this relationship was

RVP = 41.3 + (-14.9) (RMSV) $\pm$ 4.9 mm Hg.

TABLE X A

PATIENTS WITH ATRIAL SEPTAL DEFECT

PATIENTS	AGE	RVP	PA	P	RMSV & ORIENT.	ECG DIAGNOSIS
D. E.	9 10/12	20/2	16/6		.78 nl anterior	PBH
M. T.	6 2/12	28/1	28/10		.97 nl posterior	PRB RVH
B. C.	11/12	28/3	30/12		.99 incr anterior	POSS RVH
K. D.	5 2/12	32/6	24/10		.88 nl anterior	DEF RVH
A. C.	8 7/12	37/6	30/11		.50 decr posterior	POSS RVH

TABLE X B

RESULTS IN PATIENTS WITH ATRIAL SEPTAL DEFECT
ACCURACY OF ECG AND XYZ CRITERIA IN
IDENTIFYING PATIENTS WITH ELEVATED RIGHT
VENTRICULAR PRESSURES

Criteria	False Positive	Specificity (1 - fp)	False Negative	Sensitivity (1- f n)
ECG	2/3	1/3	0/2	2/2
XYZ VOLTAGE	1/3	2/3	2/2	0/2
XYZ VOLT. & ORIENTATION	1/3	2/3	2/2	0/2

Altogether, there were thirty-five patients in whom an elevated right ventricular peak systolic pressure might have been expected due to the presence of a congenital heart defect causing either a right ventricular pressure or volume overload. Those patients with a right ventricular pressure overload included thirteen with pulmonic stenosis, four with ventricular septal defect and pulmonary artery band, and six with Tetralogy of Fallot. Those in whom a right ventricular pressure overload secondary to a volume overload was possible included seven patients with a ventricular septal defect and four with an atrial septal defect.

Of these thirty-five patients, only thirty were actually found to have a right ventricular systolic pressure of 30 mm Hg or above. Twenty-nine of these thirty patients were diagnosed as having some form of right ventricular hypertrophy---either definite, probable or possible---using the standard twelve lead electrocardiogram while only eighteen had an elevated right maximal spatial vector using the XYZ. Only four of the eighteen patients with an elevated RMSV had an RMSV oriented anteriorly. On the other hand, only two of the five patients with a non elevated right ventricular pressure were incorrectly diagnosed as having an elevated right ventricular pressure by the ECG criteria as compared to four out of five by the XYZ voltage criteria.

In summary, eighteen of the patients were correctly diagnosed as either having or not having an elevated right ventricular pressure by both the ECG criteria and the XYZ voltage criteria. Four were diagnosed incorrectly using either lead system. Ten were correctly diagnosed by the ECG criteria but incorrectly diagnosed by the XYZ voltage criteria. Only one patient was diagnosed correctly by the XYZ voltage criteria , but not by the ECG criteria.

When the correlation coefficients are examined for each of the lesions in which the right ventricular pressure may be elevated, the best correlation is found for the diagnosis of isolated pulmonic stenosis: $r = 0.61$ (p less $.05$). Far less correlatiom is found in any of the other diagnoses: $r=.14$ for seven patients with ventricular septal defects, $r=-0.56$ for six patients with Tetralogy of Fallot, $r=-0.87$ for four patients with ventricular septal defect and pulmonary artery band, and $r=$

-0.48 for five patients with atrial septal defects. When all thirty-five patients are studied together, the correlation coefficient is r=.47 (p less .01) . The regression equation is RVP = 34.2 + 28.8 (RMSV) $\pm$ 31 mm Hg.

CORRELATION BETWEEN RMSV AND RIGHT VENTRICULAR PRESSURE IN PATIENTS WITH A LESION CAPABLE OF CAUSING A RIGHT VENTRICULAR PRESSURE OVERLOAD

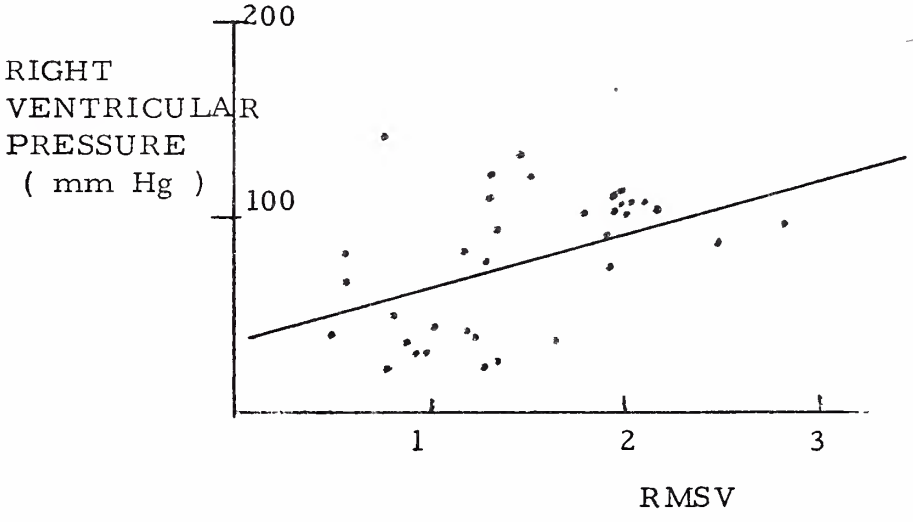

Figure 2. RMSV versus right ventricular pressure in thirty-five patients with a lesion capable of causing a right ventricular pressure overload: pulmonic stenosis, ventricular septal defect with pulmonary artery band, Tetralogy of Fallot, ventricular septal defect, or atrial septal defect. Correlation coefficient r = 0.465 (p less .01). Regression equation for relationship between RMSV and right ventricular pressure is RVP = 34.2 + 28.8 (RMSV) $\pm$ 31 mm Hg.

LEFT VENTRICULAR PRESSURE OVERLOAD PATIENTS WITH AORTIC STENOSIS

Aortic stenosis, with varying degrees of associated aortic insufficiency, was confirmed at catheterization in twelve patients ranging from 1 3/12 to 17 3/12 years of age. The peak systolic left intraventricular pressures (LVP) ranged from 120 to 200 mm Hg. In the four patients in whom left ventricular pressure was not considered elevated, the ECG incorrectly diagnosed two as being elevated (false positive rate 2/4 and specificity 2/4). The XYZ voltage and the XYZ voltage and orientation criteria were even less specific---both incorrectly diagnosing three of the four as having elevated LVP (false positive rate of 3/4 and specificity 1/4). When the finding of an increased voltage in a maximal spatial vector oriented posteriorly and in either the left or right direction (the posterior orientation XYZ criteria described in part III of Table III) was used to identify an elevated LVP, there was no change in the specificity of the XYZ lead system criteria for elevated LVP.

The third set of XYZ criteria or the posterior orientation criteria had the greatest sensitivity of all the criteria used to identify the patients who had an elevated LVP. The sensitivity of the posterior orientation criteria was 8/8, compared to 5/8 for both the XYZ voltage and the XYZ voltage and orientation criteria and 6/8 for the ECG criteria. Only five of the eight patients with an elevated LVP did have a LMSV of increased (elevated) voltage oriented posteriorly.

But while none of the patients with aortic stenosis had an elevated RVP, of the eight who had an elevated LVP, five had an increased RMSV directed posteriorly, one had a normal RMSV directed posteriorly, and two had RMSV directed anteriorly which were of normal magnitude. The implication is that the presence of an increased RMSV oriented posteriorly occurs as frequently as does an increased LMSV oriented posteriorly in aortic stenosis. There were no instances of an increased RMSV oriented posteriorly not being associated with an elevated LVP.

The correlation coefficient for the relationship between the LVP and LMSV was 0.32 ($p > .1$). The regression equation for this relationship was LVP =130 + 8.5(LMSV) $\pm$ 26 mm Hg.

Despite the rather poor correlation of the ventricular pressures with the voltages, the accuracy of the XYZ criteria in diagnosing elevations of left ventricular pressure was fair. Of the twelve patients studied, the ECG and the XYZ voltage criteria were both correct in four instances and both incorrect in two. The ECG criteria was correct while the XYZ was incorrect in four instances. But the XYZ was correct while the ECG was incorrect in two.

TABLE XI A PATIENTS WITH AORTIC STENOSIS

PATIENT	ASSOCIATED LESION	AGE	LVP	LMSV & ORIENTATION *	ECG DIAGNOSIS	RMSV & ORIENTATION
L.K.	isolated	14 1/12	120/8 nl	1.35 decr	NO LVH	normal posterior
R.Ce.	AI	5 10/12	120/75 incr	2.75 incr	NO LVH	incr posterior
L.G.	AI	15 6/12	123/13 nl	3.01 incr	DEF LVH	incr anterior
S.K.	isolated	9 2/12	125/12 high nl	2.56 incr	POSS LVH	decr anterior
A.T.	isolated	13 8/12	133/13 high nl	2.50 incr	NO LVH	incr posterior
Ja. A.	membranous	5 11/12	150/4 incr	.91 decr*	PBH	incr posterior
L.Co.	AI	15 2/12	150/8 incr	1.81 nl	PBH	incr posterior
J.T.	subvalvular, s/p coarct repair	11 6/12	160/4 incr	1.71 nl*	PROB LVH	incr posterior
C.L.	isolated	15 9/12	160/5 incr	2.59 incr	POSS LVH	normal anterior
K.S.	isolated	1 3/12	185/5 incr	4.76 incr	DEF LVH	incr posterior
C.C.	isolated	17 3/12	187/7 incr	1.60 incr	NO LVH	normal posterior
R.Cd.	AI	17 1/12	200/10 incr	3.53 incr	POSS LVH	normal anterior

* Unless starred(*), all LMSV were oriented posteriorly.

TABLE XI B

RESULTS IN PATIENTS WITH AORTIC STENOSIS
ACCURACY OF ECG AND XYZ CRITERIA IN
IDENTIFYING PATIENTS WITH ELEVATED LEFT
VENTRICULAR PRESSURE

Criteria	False Positive	Specificity (1 - f p)	False Negative	Sensitivity (1 - fn)
ECG	2/4	2/4	2/8	6/8
XYZ VOLTAGE	3/4	1/4	3/8	5/8
XYZ VOLTAGE WITH LEFTWARD AND POSTERIOR ORIENTATION	3/4	1/4	3/8	5/8
XYZ VOLTAGE WITH POSTERIOR ORIENTATION ONLY	3/4	1/4	3/8	5/8

PATIENTS WITH COARCTATION OF THE AORTA

The diagnosis of coarctation of the aorta was confirmed at catheterization in nine patients ranging in age from 3 8/12 to 13 3/13 years of age. Since left ventricular systolic pressures were not available when the coarctation was demonstrated only with a levo phase film of a right ventricular contrast injection, blood pressures taken in the arms, expected to be perhaps 10 mm Hg higher than the left ventricular peak systolic pressure, were used as the basis for determining the degree of correlation between the left ventricular pressure (blood pressure) and the XYZ voltages.

In the one patient in whom the left ventricular pressure (actually blood pressure) was not elevated, none of the criteria for increased left ventricular forces diagnosed an elevated left ventricular pressure. Hence, the false positive rate was 0/1 and, by definition, the specificity of all the criteria was 1/1. Of the eight patients in whom the blood pressure (left ventricular pressure) was elevated, three were correctly identified by the

ECG and four by the XYZ posterior orientation criteria. Thus, the sensitivities for these two sets of criteria were 3/8 and 4/8, respectively. On the other hand, the XYZ voltage and the XYZ voltage and orientation criteria each identified only one of the eight patients correctly (i.e. sensitivity 1/8).

Of the seven patients in whom the XYZ voltage and the XYZ voltage and orientation criteria yielded false negative diagnoses, five had decreased to high normal LMSV's oriented posteriorly while two had anteriorly directed LMSV's: one high normal and one decreased in magnitude. In contrast, three of these seven patients had an increased RMSV oriented posteriorly, two had a posteriorly oriented non elevated RMSV, and two had decreased anteriorly oriented RMSV's.

If one excludes from consideration those of the seven false negative-by-XYZ-voltage-criteria patients who had elevated right ventricular as well as left ventricular pressures, there are two patients with normal right ventricular pressure and increased posteriorly oriented RMSV: one with a RMSV of normal magnitude directed posteriorly and one with no rightward forces and normal right ventricular systolic pressure.

Although the interplay of forces is not completely understood, the results do indicate that posterior forces, whether oriented left or right, are helpful in identifying elevated left ventricular or blood pressures.

Overall, the ECG and XYZ were correct in only one instance and both were incorrect in four instances. The ECG was correct but the XYZ incorrect in three instances and the XYZ correct but the ECG incorrect in one instance.

The correlation coefficient calculated between the blood pressure or the left ventricular systolic pressure and the LMSV was 0.43 (p more .1). The regression equation for this relationship was LVP (or blood pressure) = 123 + 13.7 (LMSV) $\pm$ 16.2 mm Hg.

TABLE XII A PATIENTS WITH COARCTATION OF THE AORTA

PATIENT	AGE	BLOOD PRESSURE	LMSV & ORIENTATION		ECG DIAGNOSIS	RVP	RMSV & ORIENTATION
R.G.	5 2/12	105/6 (LVP)high nl	1.09	decr A	NO LVH	20/	incr posterior
R.B.	6 6/12	138/80 incr	2.33	high nl A	POSS LVH	35/	incr posterior
D.G.	6 3/12	140/70 incr	1.29	decr P	NO LVH	30/3	incr posterior
C.R.	6 7/12	145/100 incr	1.50	low nl P	NO LVH	20/3	decr anterior
G.H.	13 3/12	150/97 incr	2.30	high nl P	POSS LVH	26/2	normal posterior
T.H.	12 7/12	150/100 incr	1.92	normal P	PBH	34/9	decr anterior
K.K.	3 8/12	160/105 incr	.82	decr A	NO LVH	33/	decr posterior
J.B.	5 3/12	165/11 (LVP) incr	2.11	nl P	NO LVH	20/	incr posterior
E.M.	5 2/12	170/85 incr	2.49	incr P	RVCD	40/7	decr anterior

TABLE XII B

RESULTS IN PATIENTS WITH COARCTATION OF THE AORTA

ACCURACY OF ECG AND XYZ CRITERIA IN IDENTIFYING PATIENTS WITH ELEVATED LEFT VENTRICULAR PRESSURE

Criteria	False Positive	Specificity	False Negative	Sensitivity
ECG	0/1	1/1	5/8	3/8
XYZ VOLT.	0/1	1/1	7/8	1/8
XYZ VOLT. WITH LEFTWARD AND POSTERIOR ORIENTATION	0/1	1/1	7/8	1/8
XYZ VOLT. WITH POSTERIOR ORIENTATION ONLY	0/1	1/1	4/8	4/8

In summary, there were twenty-one patients who carried a diagnosis in which a pressure overload of the left ventricle was possible. Of these twenty-one patients, only sixteen had a left ventricular pressure (or, in the case of coarctation of the aorta, blood pressure) which was considered abnormally elevated. ECG criteria correctly identified nine of these sixteen patients while an abnormally increased LMSV was found in only six of these sixteen patients. On the other hand, ECG criteria applied to the five patients who did not have an elevated left ventricular pressure resulted in a false positive rate of 2/5 while the XYZ voltage criteria resulted in a false positive rate of 3/5.

Twelve of the sixteen patients with elevated left ventricular pressure were identified by either a left or right maximal spatial vector which was increased in magnitude and oriented

posteriorly compared to only six out of sixteen identified by elevations of LMSV oriented either anteriorly or posteriorly. There was no associated rise in the false positive rate when elevations of maximal spatial vectors oriented posteriorly and with either left or right orientation were used to diagnose elevations in left ventricular pressure.

Furthermore, of the twelve patients (four with diagnoses of coarctation of the aorta and eight with aortic stenosis) who had an elevated left ventricular pressure but no increase in right ventricular pressure, seven (two coarctations and five aortic stenoses) had an elevated RMSV oriented posteriorly. These results reveal that a pure elevation in left ventricular pressure (i.e. with no associated right ventricular pressure overload) results in an increased posterior maximal spatial vector oriented rightwards in over half (7/12) the number of cases, instead of leftwards as one might expect.

In these twenty-one cases of left ventricular pressure overload, the ECG and XYZ voltage criteria were correct in five instances and both were incorrect in six instances. The ECG was correct and the XYZ incorrect in seven instances and the XYZ correct and the ECG incorrect in three.

The correlation coefficient between the LVP and LMSV in all twenty-one cases taken together was $r = 0.35$ (p more .1). The regression equation for this relationship was

$LVP = 130 + 9.1\ (LMSV) \pm 22$ mm Hg.

CORRELATION BETWEEN LMSV AND LEFT VENTRICULAR PRESSURE OR BLOOD PRESSURE* IN THE ARMS OF PATIENTS WITH A LESION CAPABLE OF CAUSING A LEFT VENTRICULAR PRESSURE OVERLOAD

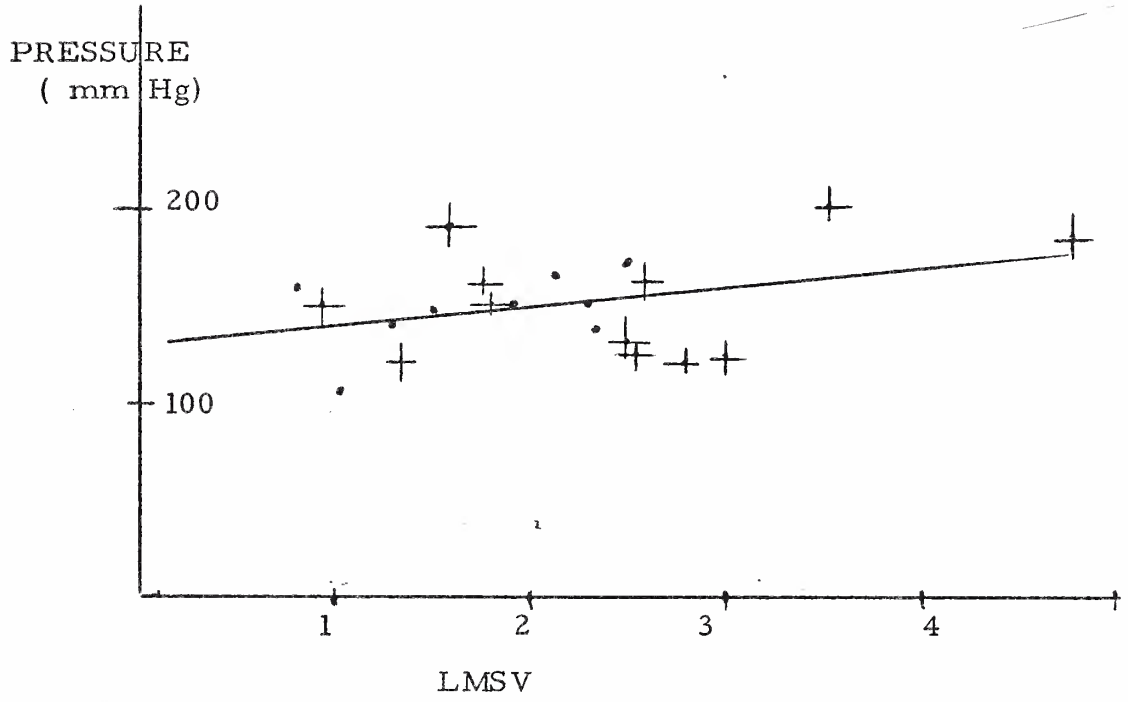

Figure 3. * LMSV versus left ventricular pressure (crosses for the twelve patients with aortic stenosis) or blood pressure in the arms (dots for the nine patients with coarctation of the aorta). Correlation coefficient r = 0.35 (p more .1). Regression equation is : (LVP or BP) = 130 + 9. . (LMSV) $\pm$22mm Hg.

LEFT VENTRICULAR VOLUME OVERLOAD

PATIENTS WITH PATENT DUCTUS ARTERIOSUS

The diagnosis of patent ductus arteriosus (PDA) was confirmed at catheterization in eighteen patients ranging from 10/12 to 13 0/12 years of age and with shunts ranging from a Qp/Qs of 1.1 to 2.1. Since there is no distal mixing chamber from which one can sample to determine the degree of mixing of systemic and pulmonary blood in the great vessels, Qp/Qs is not an ideal parameter with which to quantitate the shunt of a PDA. Nevertheless, the ECG and XYZ findings can be compared using the calculated Qp/Qs as a rough guide to hemodynamic function.

The eighteen patients with a patent ductus arteriosus for whom Qp/Qs was calculated or estimated during angiography at catheterization were divided into two groups: those with Qp/Qs over 1.5 and those with Qp/Qs not over 1.5. The ECG criteria were found to be extremely sensitive in their ability to identify ECG's of patients with Qp/Qs over 1.5 (sensitivity was 4/4) but not very specific (false positive rate was 9/14). On the other hand, the XYZ voltage criteria were more specific (false positive rate was 5/14) but less sensitive (sensitivity was 2/4). Seven patients had Qp/Qs values in the range correctly predicted by both the ECG and XYZ voltage criteria. Six had Qp/Qs values incorrectly predicted by both lead criteria. In four instances, the ECG criteria was incorrect while the XYZ voltage criteria was correct. And in one instance, the XYZ voltage criteria was incorrect while the ECG was correct.

The correlation coefficient for the relationship between the Qp/Qs and LMSV voltage values was 0.425 (p more 0.1). The regression equation for this relationship was $Qp/Qs = .99 + (.14)\ (LMSV) \pm .26$. In contrast, the correlation coefficient for the relationship between the RMSV and the right ventricular pressure in this left ventricular volume loaded lesion was -0.21 for the fourteen patients.

TABLE XIII A PATIENTS WITH PATENT DUCTUS ARTERIOSUS

PATIENT	AGE	LVP	Qp/Qs	LMSV & ORIENTATION	ECG DIAGNOSIS
C. L.	7 6/12	100/13 nl	1.1	2.33 high nl posterior	POSS LVH
J. B.	8 6/12	120/4 nl	1.1	1.28 decr posterior	NO LVH
S. Sy.	13 0/12	110/10 nl	1.1	1.71 nl posterior	NO LVH
D. S.	4 0/12	100/60 nl	small	1.71 low nl anterior	PBH
M. B.	10/12	105/3 incr	1.1	2.73 incr anterior	POSS LVH
S. H.	2 4/12	100/9 incr	small	2.22 normal anterior	NO LVH
E. O.	1 1/12	100/7 incr	small	2.65 incr anterior	POSS LVH
T. C.	8 11/12	130/15 incr	1.1	2.12 normal posterior	NO LVH
G. K.	1 3/12	115/15 incr	small	2.51 incr posterior	PBH
L. H.	5 4/12	87/6 nl	1.3	3.58 incr posterior	POSS LVH
S. W.	3 8/12	120/10 incr	1.3	2.19 normal posterior	POSS LVH
S. Wo.	4 11/12	115/60 incr	1.3	2.86 incr posterior	POSS LVH
S. Sc.	5 1/12	110/9 nl	1.4	1.40 decr posterior	POSS LVH
J. W.	3 1/12	112/8 nl	1.5	1.05 decr anterior	NO LVH
L. P.	4 6/12	130/10 incr	mod	2.20 normal posterior	POSS LVH
M. M.	3 5/12	120/80 incr	mod	1.53 decr posterior	DEF LVH
V. H.	6 4/12	135/87 incr	mod	3.42 incr posterior	POSS LVH
L. J.	5 1/12	110/60 nl	2.1	3.84 incr posterior	PROB LVH

TABLE XIII B

RESULTS IN PATIENTS WITH PATENT DUCTUS ARTERIOSUS
ACCURACY OF ECG AND XYZ CRITERIA IN
IDENTIFYING PATIENTS WITH Qp/Qs OVER 1.5

Criteria	False Positive	Specificity (1 - f p)	False Negative	Sensitivity (1 - f n)
ECG	9/14	5/14	0/4	4/4
XYZ VOLT.	5/14	9/14	2/4	2/4
XYZ VOLT. & ORIENTATION	4/14	10/14	2/4	2/4

LEFT VENTRICULAR VOLUME OVERLOAD (cont.)

PATIENTS WITH VENTRICULAR SEPTAL DEFECT

The diagnosis of ventricular septal defect was made at catheterization in seven patients. Qp/Qs values ranged from 1.1 to 3.0. When the patients, ranging in age from 10/12 to 12 7/12 years of age, were arbitrarily divided into two groups, those with Qp/Qs over 1.5 and those not over 1.5, it was found that the sensitivity of the three sets of criteria were equally sensitive in detecting a Qp/Qs over 1.5. In each instance, two of the four patients with Qp/Qs over 1.5 were identified by the ECG, XYZ voltage and XYZ voltage and orientation criteria. However, the ECG was less specific and had a false positive rate of 2/3 while neither the XYZ voltage nor the XYZ voltage and orientation criteria had any false positives.

While the ECG and XYZ voltage criteria were both correct in two instances; the ECG was correct and the XYZ voltages incorrect in two instances; and in three instances,

the ECG was incorrect and the XYZ correct.

The correlation coefficient between the Qp/Qs and the LMSV was r =0.76 ($p < .1$). The regression equation for this relationship was $Qp/Qs = .30 + .87(LMSV) \pm .50$.

TABLE XIV A

PATIENTS WITH VENTRICULAR SEPTAL DEFECT

PATIENT	AGE	LVP	Qp/Qs	LMSV & ORIENTATION	ECG DIAGNOSIS
B. Ma.	7 4/12	110/6 nl	1.1	1.81 nl posterior	NO LVH
R. C.	12 7/12	130/18 nl	1.2	1.02 decr anterior	PROB LVH(BVH
B. Mo.	10 7/12	110/17 nl	1.4	1.90 nl posterior	NO LVH
L. M.	10/12	85/10 nl	2.2	1.35 decr anterior	PROB LVH(BVH
R. R.	3 11/12	100/9 nl	2.7	3.01 incr anterior	NO LVH
J. A.	11/12	110/15 incr	2.9	2.80 incr anterior	NO LVH
K. S.	4 6/12	88/5 nl	3.0	2.79 incr anterior	POSS LVH(BVH

TABLE XIV B

RESULTS IN PATIENTS WITH VENTRICULAR SEPTAL DEFECT
ACCURACY OF ECG AND XYZ CRITERIA IN IDENTIFYING
PATIENTS WITH Qp/Qs OVER 1.5

Criteria	False Positive	Specificity (1 - f p)	False Negative	Sensitivity (1 - f n)
ECG	2/3	1/3	2/4	2/4
XYZ VOLT.	0/3	2/3	2/4	2/4
XYZ VOLT. & ORIENTATION	0/3	2/3	2/4	2/4

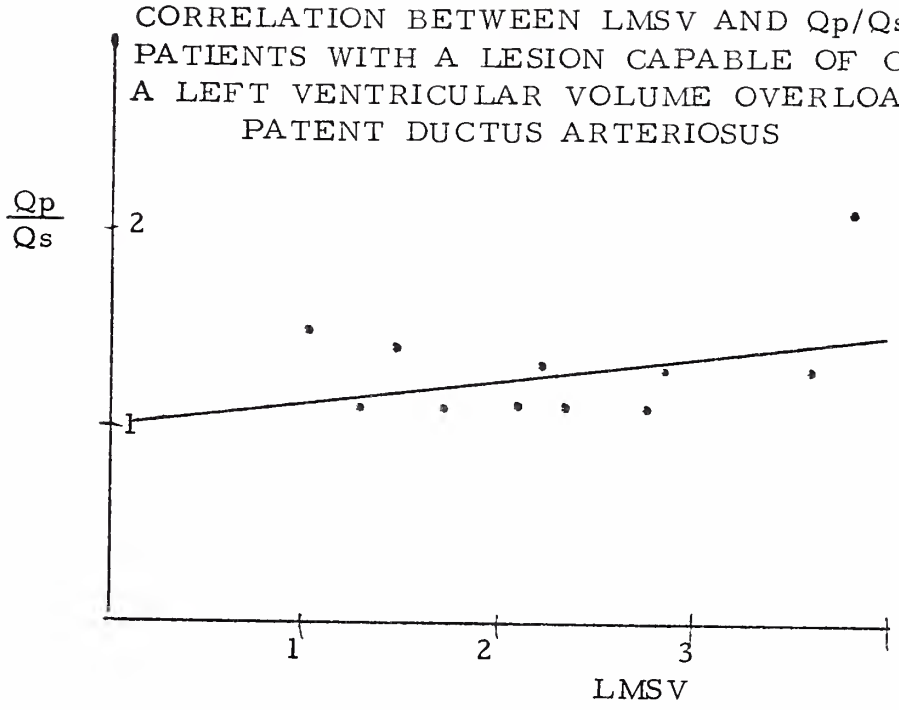

Figure 4. LMSV versus Qp/Qs in eleven patients with patent ductus arteriosus. Correlation coefficient r = .43 (p more 0.1). Qp/Qs = .99 +(.14) (LMSV) $\pm$ 0.26.

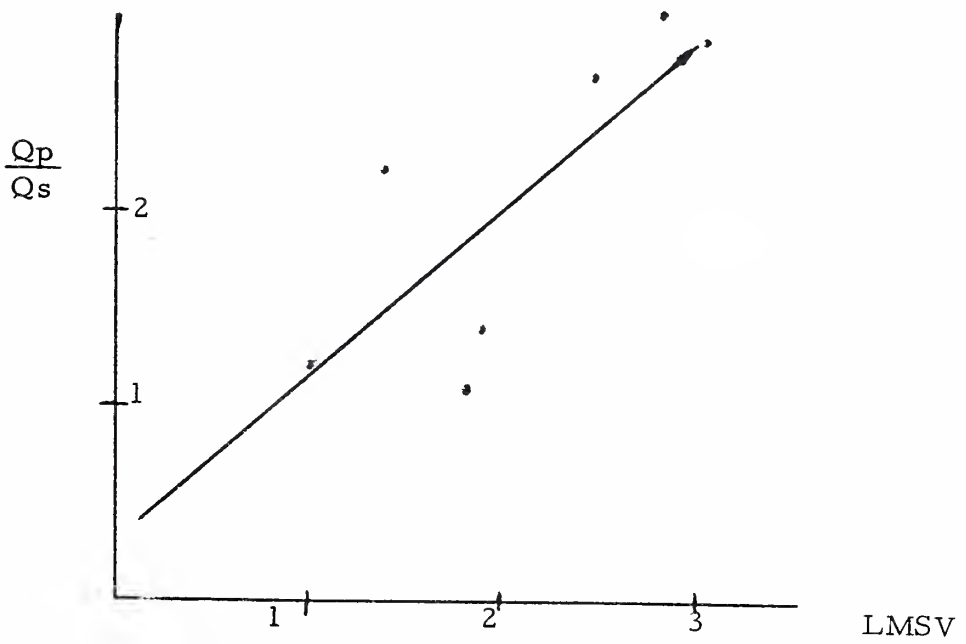

Figure 5. LMSV versus Qp/Qs in seven patients with ventricular septal defect. Correlation coefficient r = .76 (p less 0.1). Qp/Qs = .30 + .87 (LMSV) $\pm$ .50.

When these twenty-five patients with a diagnosis of ventricular septal defect or patent ductus arteriosus were combined, the correlation coefficient for the relationship between the degree of left ventricular volume overload as represented by Qp/Qs and the LMSV was r = 0.386 (p less 0.1). The regression equation for this relationship was Qp/Qs =0.94 + (0.31) (LMSV) $\pm$ 0.56.

Although the Qp/Qs approximation of the shunt in a patent ductus arteriosus is not optimal, the results of the LVH criteria applied to these twenty-five patients indicated that the ECG was more sensitive in identifying situations of Qp/Qs over 1.5, while the XYZ criteria was less sensitive although more specific. Thus, the sensitivity of the ECG criteria in identifying the eight individuals who had a Qp/Qs over 1.5 was 6/8; the sensitivity of the XYZ voltage criteria was 4/8; and the sensitivity of the XYZ voltage and orientation criteria was also 4/8. On the other hand, the false positive rate for the ECG was 11/17; for the XYZ voltage criteria, it was 5/17; and, for the XYZ voltage and orientation criteria, it was 4/17.

CORRELATION BETWEEN LMSV AND Qp/Qs IN PATIENTS WITH LESION CAPABLE OF CAUSING A LEFT VENTRICULAR VOLUME OVERLOAD

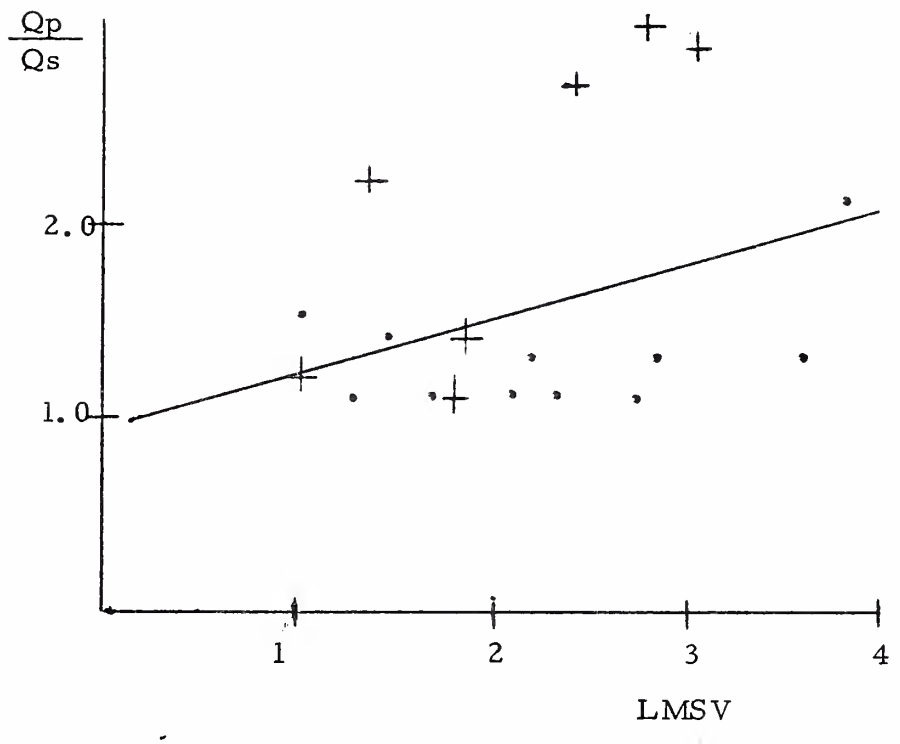

Figure 6. LMSV versus Qp/Qs in patients with possible left ventricular volume overload. (Cross: 7 patients with ventricular septal defect. Dot: 18 patients with patent ductus arteriosus.) Correlation coefficient r= 0.37 (p less 0.1). Regression equation for relationship between LMSV and Qp/Qs is Qp/Qs = 0.94 + (.31) (LMSV) $\pm$ 0.56.

RIGHT VENTRICULAR VOLUME OVERLOAD

PATIENTS WITH ATRIAL SEPTAL DEFECT

There were five patients with an atrial septal defect, the classical example of a right ventricular volume overload. Their ages ranged from 11/12 to 9 10/12 years of age. The Qp/Qs values ranged from 1.6 to 2.2. When the patients were arbitrarily divided into two groups, those with Qp/Qs over 2 and those 2 or less, it was found that the ECG had a false positive rate of 2/3 and specificity 1/3 in identifying shunts having a Qp/Qs over 2. The XYZ voltage and the XYZ voltage and orientation criteria had a higher specificity (3/3). On the other hand, as would be expected, the XYZ had a lower sensitivity (1/2) than the ECG , whether the XYZ voltage criteria or the voltage and orientation criteria was used.

The correlation coefficient for the Qp/Qs and RMSV relationship was r = 0.79 (p more .1). The regression equation for this relationship was Qp/Qs = 1.1 + 1.0 (RMSV) $\pm$ .14.

TABLE XV A

PATIENTS WITH ATRIAL SEPTAL DEFECT

PATIENT	AGE	RVP	$\frac{Qp}{Qs}$	RMSV & ORIENTATION	ECG DIAGNOSIS
D.E.	9 10/12	20/2	1.6	.78 nl anterior	PBH
A.C.	8 7/12	37/6	1.7	.50 decr posterior	POSS RVH
K.D.	5 2/12	32/6	2.0	.88 nl anterior	DEF RVH
M.T.	6 2/12	28/1	2.1	.97 nl posterior	PROB RVH
B.C.	11/12	28/3	2.2	.99 incr anterior	POSS RVH

TABLE XV B

RESULTS IN PATIENTS WITH ATRIAL SEPTAL DEFECT
ACCURACY OF ECG AND XYZ CRITERIA IN
IDENTIFYING PATIENTS WITH Qp/Qs
OVER 1.5

Criteria	False Positive	Specificity (1 - f p)	False Negative	Sensitivity (1 - f n)
ECG	2/3	1/3	0/2	2/2
XYZ VOLT.	0/3	3/3	1/2	1/2
XYZ VOLT. AND ORIENTATION	0/3	3/3	1/2	1/2

CORRELATION BETWEEN RMSV AND Qp/QS IN PATIENTS WITH LESION CAPABLE OF CAUSING RIGHT VENTRICULAR VOLUME OVERLOAD

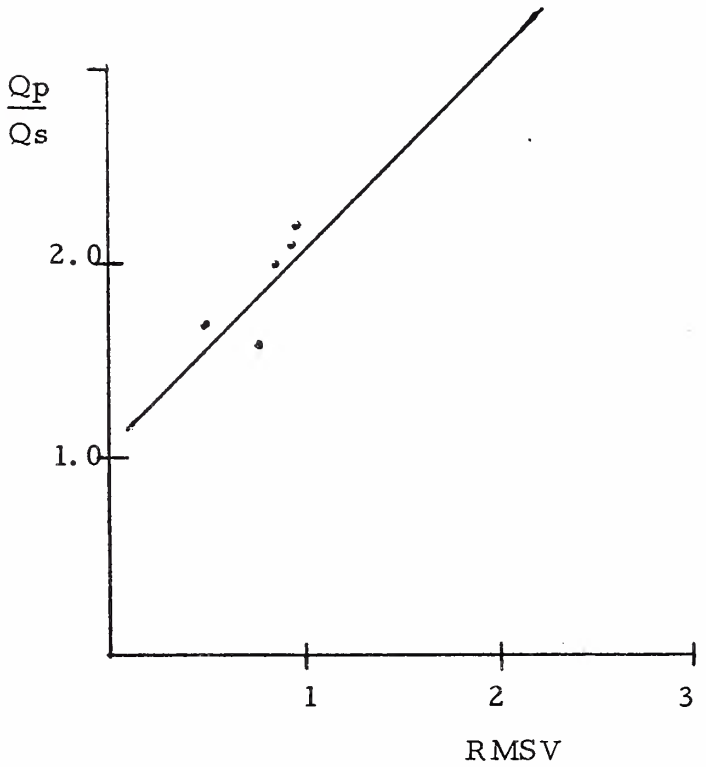

Figure 7. RMSV versus Qp/Qs in five patients with a lesion capable of causing right ventricular volume overload: atrial septal defect. Correlation coefficient r = 0.79 (p more 0.1). Regression equation for this relationship between Qp/Qs and RMSV is Qp/Qs = 1.08 +1.02(RMSV) $\pm$ .14.

TABLE XVI

DISTRIBUTION OF MAXIMAL SPATIAL VECTORS BY ORIENTATION AND VENTRICULAR PRESSURE ELEVATION

	NORMAL RVP	INCREASED RVP	INCREASED LVP	NORMAL LVP
RIGHT ANTERIOR	10	15	-	-
RIGHT POSTERIOR	22	27	-	-
LEFT POSTERIOR	-	-	25	19
LEFT ANTERIOR	-	-	13	17

Table XVI highlights some of the observations which can be made regarding the anterior-posterior direction of maximal spatial voltages which were associated with elevations in peak systolic intraventricular pressures. Of the seventy-four patients included in this study, independent of anatomic diagnosis, 42 had a right ventricular peak systolic pressure above 30 mm Hg which was considered elevated and abnormal while 32 had a right ventricular pressure not over 30 mm Hg and which was considered normal. Of the 42 patients with an elevated peak systolic intraventricular pressure, 27 had a right maximal spatial vector directed posteriorly whether increased in magnitude or not, while only 15 of the 42 patients had a right maximal spatial vector directed anteriorly. This distribution was similar to that of patients with normal right ventricular pressure, among whom there were 22 with a right maximal spatial vector directed posteriorly and 10 anteriorly.

The results of Table XVI show an almost identical distribution of anterior-posterior forces among the left maximal spatial vectors, regardless of whether or not the left ventricular peak systolic pressure was elevated. Of the 38 patients with an elevated left ventricular systolic pressure, 25 had a left maximal spatial vector directed posteriorly, while 13 had a left maximal spatial vector directed anteriorly. This was similar to the distribution of the left maximal spatial vectors of the 36 patients with normal left ventricular pressures, among

whom 19 had left maximal spatial vectors directed posteriorly and 17 directed anteriorly.

The correlation coefficients for the relationship between the maximal spatial voltages and the peak systolic intraventricular pressures was also calculated in each of the two groups of patients with quantifiable volume overloads: atrial septal defect and ventricular septal defect. In atrial septal defect, r for the voltage-pressure correlation was -0.48 ($p > 0.1$) compared to r = 0.79 ($p > 0.1$) for the voltage-Qp/Qs correlation coefficient. In the group of patients with a ventricular septal defect, the r calculated for the voltage-left ventricular pressure correlation was 0.142 ($p > 0.1$) compared to r = 0.75 ($p > 0.1$) for the voltage-Qp/Qs relationship.

When 45 patients, independent of lesion, were randomly selected, the correlation in this group between the right maximal spatial voltage and the right ventricular peak systolic pressure was 0.4 ($p < 0.5$) , while the correlation between the left maximal spatial vector voltage and the left peak systolic pressure was 0.1 ($p > 0.1$).

IV. DISCUSSION

The major factor in determining whether or not the standard twelve lead electrocardiogram (ECG) can be replaced with a Frank scalar XYZ is the ability of each lead system to aid the physician in evaluating cardiac function. While the XYZ lead system is thought to have theoretical advantages over the ECG, there have been no data to show whether or not the more realistic design and higher correlations found between the XYZ electrocardiogram and hemodynamic parameters can be as effective as the ECG in diagnosing cardiac abnormalities in pediatric patients.

In this study of 74 patients with isolated congenital heart defects documented by subsequent catheterization but unknown to the author at the time the ECG's and XYZ's were interpreted, the results show that the voltage and orientation of increased XYZ forces provide different and generally less sensitive and specific information than does the ECG. Of the three sets of criteria which were designed to assess the ability of the XYZ to identify ventricular overloads documented by the catheterization, only one proved possibly more advantageous than conventional ECG voltage and orientation criteria. In addition, the correlation coefficients calculated for the relationship between the magnitude of the maximal spatial voltages and either the peak systolic intraventricular pressures or the volume overloads as expressed by Qp/Qs were low and differed from those in the well known work of Ellison and Restiaux (1972).

The comparison of the specificities and sensitivities of the XYZ and ECG in identifying the ventricular overloads reveals

three especially useful insights into the relative value of the voltage and orientation requirements for making the diagnosis of an overload. First, without exception, ECG criteria for right and left ventricular hypertrophy were more accurate than any combination of the respective (right or left chamber) maximal spatial voltage with or without the anterior-posterior orientation requirements. Second, right-left maximal spatial voltage requirements were more reliable without the anterior-posterior requirements. Third, cardiac lesions involving a left ventricular pressure overload may be associated with a higher frequency of rightward, posteriorly maximal spatial voltages than commonly expected.

The very nature of these three conclusions permits one to speculate why the ECG seems to be a more useful lead system and how the XYZ may be developed into one that is as accurate.

It is not difficult to explain why the ECG seems to be a more sensitive lead system. As Abildskov (1958) and Pipberger(1961) have shown, the simple availability of more ECG leads with which to find positive signs of increased ventricular overload increases the possibility of a positive identification. Moreover, in this particular series of patients, all of whom were felt to have some cardiac disease or they would not have been catheterized and included in this study, the larger number of ECG minor criteria used made the ECG more sensitive in the identification of ventricular overloads. The lack of specificity implied by applying a larger number of criteria was not as severe a disadvantage as it might have been in a group of patients who did not warrant catheterization.

Since, as Pipberger (1961) has shown, the amount of information contained in the XYZ is the same as in the ECG, one wonders whether additional criteria ---e.g. examination of the 10 and 20 millisecond vectors or scalar as well as spatial magnitudes---might have been of diagnostic value. As Reeves (1970) pointed out, there is no such thing as criteria which are both specific and sensitive. Increase the number of criteria and one is bound to have more sensitive criteria. Thus, the compression of information into three as opposed to twelve leads may have reduced the sensitivity of the XYZ criteria in detecting ventricular overload.

Why the XYZ criteria performed better when the anterior-posterior restrictions on the criteria for increased left or right ventricular forces were dropped is not easy to explain. Generally, one expects to find an increased right maximal spatial voltage oriented anteriorly in right ventricular pressure overloads and an increased left maximal spatial voltage oriented posteriorly in left ventricular pressure overloads. Yet, as Table XVI (see page 77) shows, a larger percentage (27/42) of the patients with elevated right ventricular pressures had a right maximal spatial voltage directed posteriorly. At the same time, thirteen of the thirty-eight patients with an elevated left ventricular pressure had a left maximal spatial vector which was directed anteriorly rather than posteriorly.

The concept that increased anterior and rightward forces imply right ventricular overload and that increased posterior and leftward forces imply left ventricular overload is too well established to be disputed by the just-mentioned data

of Table XVI. Rather, the more reasonable explanation may be that the use of the direction and voltage of the maximal spatial vector as ideal parameters by which to characterize ventricular overloads is not correct. Guntheroth (1965) has criticized the maximal spatial voltage for not lending appreciation to the "fatness" of electromotive forces. Without any doubt, a maximal spatial voltage represents the direction of forces at only one moment in time, not the relative preponderance of forces over a period of time. Hence, Hugenholtz and Gamboa"s suggestion of using a summed vector which totals the voltages for the several millseconds before as well as after the time of the maximal spatial vector may be a better parameter to characterize the degree and site of the overload.

The confusion which exists over the interpretation of the left-right direction of posteriorly directed terminal forces in the ECG of patients with left ventricular p ressure overloads extended to the interpretation of the left-right forces in the XYZ's of patients with elevated left ventricular pressure. In this study, if one disregarded the left-right orientation of the maximal spatial voltages and permitted any abnormally increased posteriorly oriented maximal spatial voltage to identify a left ventricular overload, as was done in the third set of XYZ criteria, then this (the third) set of XYZ criteria is more sensitive than the ECG in detecting elevated left ventricular pressure. In the patients of this study who had a pure elevation of left ventricular pressure and an abnormally increased posteriorly oriented maximal spatial voltage, over half (7/12) of the increased posteriorly oriented maximal spatial voltages pointed rightwards. All of these patients had catheterization confirmed coarctation of the aorta or aortic

stenosis, a diagnosis which Metianu et al (1953) and others, including Liebman (1968) have associated with a significant frequency of right bundle branch block. The terminal rightward forces may be either real right bundle branch block or represent an electrocardiographic sign of elevated left ventricular pressure which simulates right bundle branch block. Clearly, the interpretation and significance of electrocardiographic patterns recorded with both XYZ and ECG leads remains incompletely understood.

Aside from the potential usefulness of XYZ criteria in distinguishing abnormal electrocardiograms from normal, correlation coefficients were calculated to determine how useful the voltages were as a predictor of either peak systolic intraventricular pressure or the volume overload shunt lesions. The results afforded five conclusions regarding the level of correlation between XYZ maximal spatial voltage magnitudes and hemodynamic parameters expressing ventricular overloading.

First, whereas Ellison and Restiaux (1972) had previously reported comparable coefficients of correlation between the right and left peak systolic intraventricular pressures, a much higher correlation coefficient ($r=.61$, p less $.05$) was found for the peak systolic pressures in pulmonic stenosis and the magnitude of the right maximal spatial voltage than between the left ventricular systolic pressures and the left maximal spatial voltage ($r= 0.43$, p more 0.1). Although our results seem at variance with those of Hugenholtz and Gamboa (1964) as well, it is of relevance that

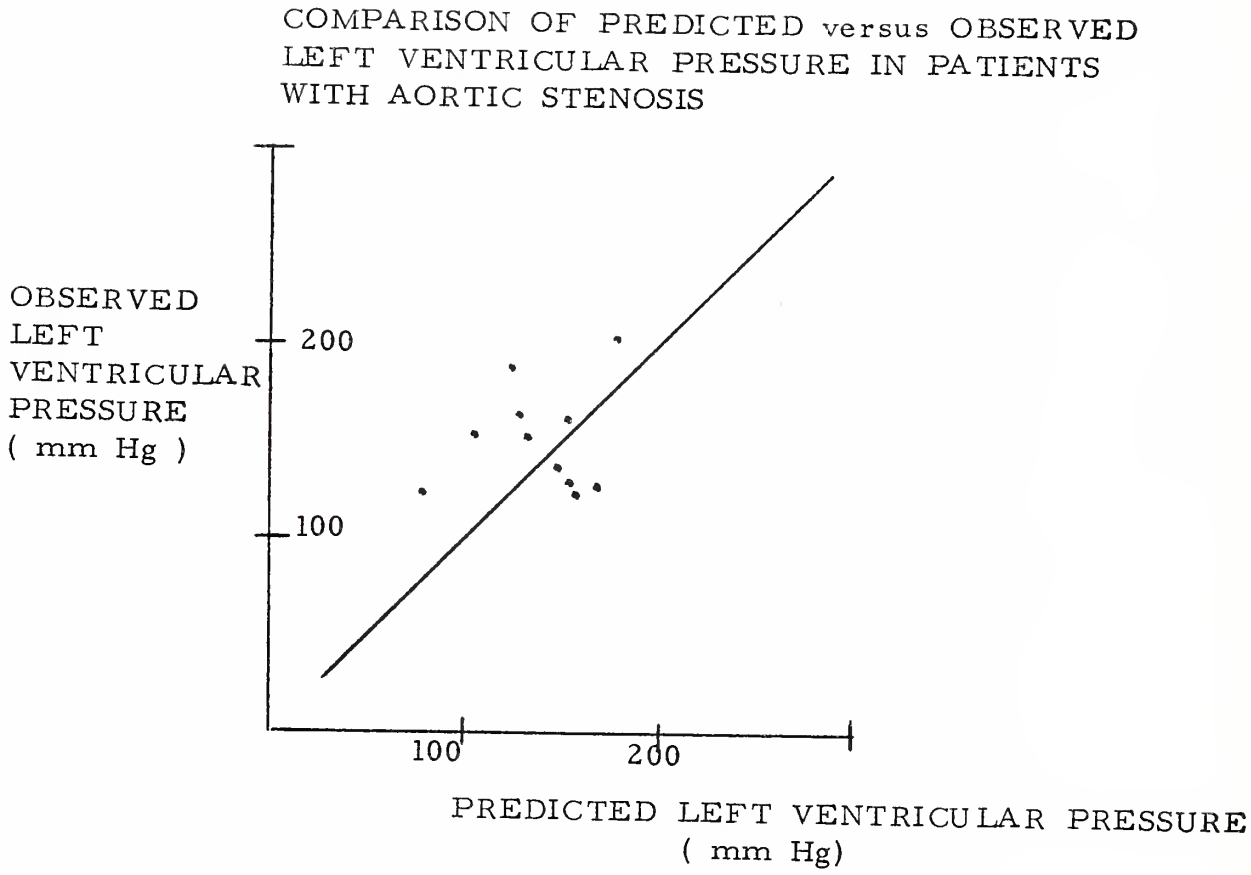

Figure 8. When the observed left ventricular pressures of the eleven aortic stenosis patients in this study were compared to the left ventricular pressures predicted from the measured LMSV's, using the regression equation for patients with aortic stenosis in the 1972 text of Ellison and Restiaux, observed values were found to be generally higher than the predicted values.

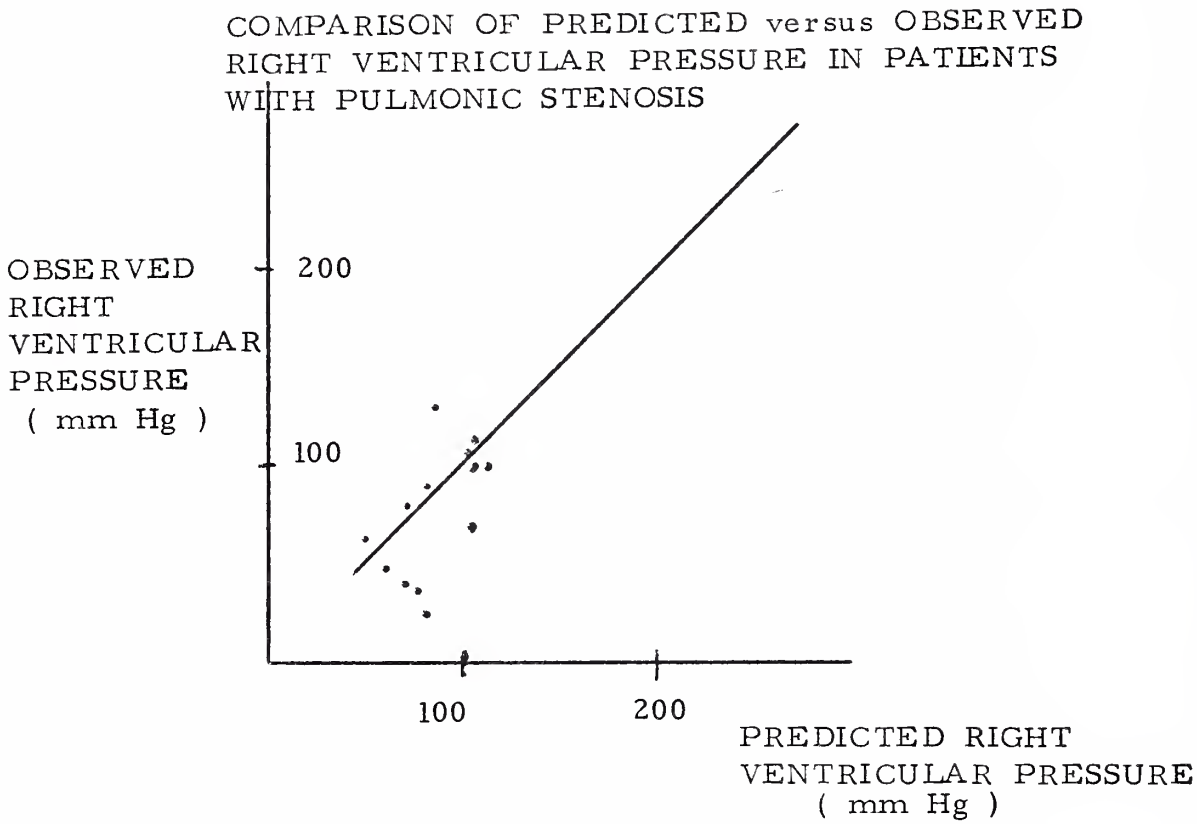

Figure 9.

When the observed right ventricular pressures of the 13 PS patients in this study were compared to the right ventricular pressures predicted from the measured RMSV's, using the regression equation for patients with pulmonic stenosis in the 1972 text of Ellison & Restiaux, observed values were found to be generally lower than the predicted values.

the ECG correlation of right ventricular systolic pressure and the height of the tallest R wave in the right precordial leads (Cayler et al, 1958) of patients with pulmonic stenosis has been recognized, while such comparably high correlations have not been published for aortic stenosis in the ECG literature.

To see if patients with ventricular pressure overloads in this study were representative of the patients in the series of Ellison and Restiaux (1972), the regression equations of Ellison and Restiaux for calculating the peak systolic left and right intraventricular pressure from the left and right maximal spatial voltages were applied to our data. As shown in Figure 8, in patients with aortic stenosis, the predicted pressures were lower than those actually observed and there was no significant increase in predictive power with higher predicted pressures. In pulmonic stenosis (see figure 9), the observed values were found to be lower than the predicted pressures and the correlations did not improve with increased predicted pressure. Hence, there may be serious doubts as to the validity of the concept of using XYZ to predict intraventricular pressure.

Second, the correlation between the maximal spatial voltage and Qp/Qs---a parameter expressing the severity of volume overloading---in a volume overloaded ventricle may be as high as the correlation between the maximal spatial voltage and peak systolic intraventricular pressure in a pressure overloaded ventricle. For example, the correlation coefficient between the right maximal spatial voltage and Qp/Qs was 0.7 in patients with patent ductus

arteriosus, which compares favorably with the coefficients calculated with pressure overloaded ventricles.

As Hugenholtz and Gamboa (1964) have pointed out, the correlation between maximal spatial voltage and peak systolic intraventricular pressure is expected to be lower than the correlation between the maximal spatial voltage and a parameter expressing the degree of volume overload in a volume overloaded ventricle, since pressure elevations are not necessarily associated with increasing severity of the volume overload. A comparison of the correlation coefficients for the voltage-pressure and voltage-Qp/Qs relationships in patients with isolated ventricular septal and isolated atrial septal defects in this study confirms this expectation.

Third, the correlation coefficient between the XYZ electrocardiographic voltages and a right ventricular volume overload (atrial septal defect) was higher than the correlation between the electrocardiographic voltages and a left ventricular volume overload (ventricular septal defect). Although the number of patients studied was small, this result runs contrary to the general expectation that volume overloads of the left ventricle are more likely to express themselves with increased electrocardiographic forces than are right ventricular volume overloads. On the other hand, increased right maximal spatial voltages may be a reflection of elevated right ventricular and pulmonary artery pressures secondary to increased volume flow to the right ventricle across a left to right shunt.

Fourth, the correlation between maximal spatial voltages and pressures is much worse in a mixed population of lesions than it is when correlations are calculated for

a population with a single isolated lesion. This is theoretically logical since the electrocardiographic morphology is dependent on the special geometry and hemodynamic condition of a given lesion. When one blindly correlates two parameters, one electrocardiographic and one hemodynamic, without pre-picking the hemodynamic parameter which is representative of that lesion the correlation cannot be expected to be high.

The implication of this finding is that while the XYZ may not be useful in assessing intraventricular pressures in an unknown population, once the anatomic diagnosis is established, the XYZ may be useful in managing and estimating the severity of the patient's lesion.

Fifth, the correlations between the maximal spatial vector magnitudes and the hemodynamic parameters are inadequate for the prediction of hemodynamic function in clinical situations. The correlations are not unexpectedly poor since the physiologic basis of the electrocardiographic patterns are not completely understood and the techniques and apparatus for measuring the electromotive forces of the heart with complete fidelity are not available. Although empirically the corrected leads seem to increase the correlation, it is not certain whether this is a theoretically sound finding. For example, since it is known that significant differences have been found in the electrocardiogram of black as opposed to white patients (Walker and Post, 1961; Traywick et al, 1973) , what would be the result of grouping patients studied by geographic origin, race, socio-economic strata, height, weight, or other anthropometric indices? Given the "love at first sight effect in research," as described by Pipberger (1968), how statistically valid are the limited numbers of patients which have

been studied in the often quoted series? What is the role of variation in lead placement in causing the lack of correlation?

Answers to these questions don't exist but are necessary to explain how the electrocardiographic findings are related to the hemodynamic parameters. At the moment, there is great confusion among electrocardiographers over precisely what constitutes criteria for ventricular hypertrophy because studies of normal electrocardiographic values in large numbers of patients appropriately categorized by various anthropomorphic indices have not been done. Ideally, as Spodick and others have pointed out, electrocardiography in the twentieth century should no longer be based on experience in subjective pattern interpretation, but on a definite sequence of objective maneuvers which can be performed by non-physicians as well (Craige, 1974). The work of Ellison and Restiaux (1972) suggesting high, direct correlations between electrocardiographic parameters and peak systolic intraventricular pressures and other hemodynamic parameters is not well supported in this study. But such efforts are necessary to the development of the electrocardiogram, using either XYZ or ECG lead systems, as an objective laboratory test which can stand on its own.

V. SUMMARY & CONCLUSIONS

The basis for using XYZ or the Frank scalar electrocardiogram in assessing the severity of loading con ditions imposed on the ventricles of children with congenital heart lesions has been reviewed. High correlations have been reported between the calculated left and right maximal spatial voltages and hemodynamic parameters obtained at catheterization, such as peak intraventricular systolic pressures and the degree of volume overload as expressed by Qp/Qs.

In this series of seventy-four patients with various isolated heart lesions (pulmonic stenosis, ventricular septal defect with pulmonary artery band, Tetralogy of Fallot, ventricular septal defect, atrial septal defect, aortic stenosis, coarctation of the aorta, and patent ductus arteriosus), conventional ECG voltage and orientation criteria showed a greater sensitivity and specificity in detecting pressure or volume overloads than either XYZ voltage or XYZ voltage and orientation criteria.

Orientation criteria for the diagnosis of increased left ventricular forces decreased the sensitivity of the XYZ voltage criteria in detecting pressure or volume overloads in all categories of patients studied. XYZ voltage criteria were more sensitive than the ECG in the diagnosis of elevated left ventricular peak systolic pressures when the left-right orientation of the posteriorly oriented maximal spatial voltage was dropped.

Correlations between the severity of volume or pressure overloads and the maximal spatial voltages were highest for right ventricular pressure overloads (pulmonic stenosis:

r=.61, p less 0.05). Other volume or pressure overload and voltage correlations were lower and less confident.

Further studies with large numbers of patients and the use of computer facilities may identify additional XYZ parameters capable of greater accuracy in the detection and evaluation of abnormal hemodynamic function.

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